Transactions of The New York Academy of Sciences

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SERIES II
VOLUME 20

NEW YORK
PUBLISHED BY THE ACADEMY
1957-1958

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SER. II, VOL. 20 *November 1957* NO. 1

TRANSACTIONS
OF
THE NEW YORK ACADEMY OF SCIENCES

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Transactions of The New York Academy of Sciences, Series II, Volume 20, No. 1, November 1957.

Entered as second-class matter December 28, 1954, at the post office at New York, N. Y., under the act of August 24, 1912. This publication is distributed to members and is published monthly from November to June, inclusive, by The New York Academy of Sciences, 2 East Sixty-third Street, New York 21, N. Y.

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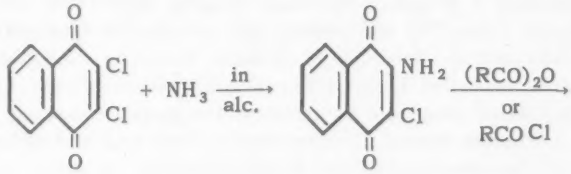
SECTION OF PHYSICS AND CHEMISTRY

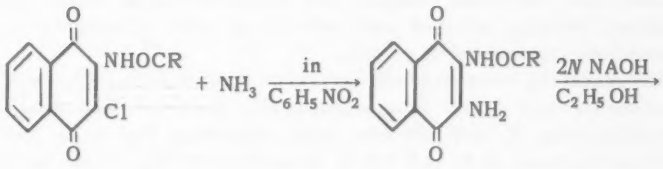
SYNTHESES OF FUSED IMIDAZOLE RING COMPOUNDS AS POTENTIAL ANTIMETABOLITES*

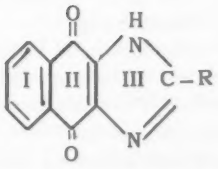
By Allan R. Day

Department of Chemistry, University of Pennsylvania, Philadelphia, Pa.

One of the objectives of the antimetabolite work at the University of Pennsylvania has been the synthesis of compounds containing two or more groups that might act as antimetabolite centers. One of the earliest attempts was the preparation of a series of imidazole derivatives of 1,4-naphthoquinone.[1] The following outline illustrates the preparation of these compounds.







R = H, CH_3, C_2H_5, n-C_3H_7, i-C_3H_7, n-C_4H_9, i-C_4H_9, n-C_5H_{11}, $(CH_3CH_2)_2$ CH, $C_6H_5CH_2$, $CH_3(CH_2)_{10}$, $CH_3(CH_2)_{14}$

The reaction of 2,3-dichloro-1,4-naphthoquinone with ammonia was carried out in refluxing ethyl alcohol solution. This reaction may also be carried out in nitrobenzene as the solvent. The acylations with acetic anhy-

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on October 1, 1957.

dride and propionic anhydride were performed at room temperature in the presence of a few drops of concentrated sulfuric acid. The remaining acylations were carried out with acyl chlorides. The 2-amino-3-chloro-1,4-naphthoquinone was suspended in the acyl chloride, and either a few drops of concentrated sulfuric acid were added, or dry hydrogen chloride was passed through the suspension for 5 min. The mixture was then refluxed for 2 to 4 hr. or until there was no further color change. The product usually crystallized on cooling. The formyl derivative could not be prepared by any of the known general methods. The chlorine atom in the 2-acylamino-3-chloro-1,4-naphthoquinone was replaced by the amino group by passing dry ammonia through its solution in nitrobenzene. The reaction can be followed by a change in color and by the deposition of ammonium chloride. After removal of the ammonium salt, the amino replacement product crystallizes on cooling. The ring closures were effected by heating the 2-acylamino-3-amino compounds in ethyl alcohol solution containing 2 *N* sodium hydroxide solution until there was no further change in color. The end product was precipitated by acidifying the concentrated solution. According to *Chemical Abstracts* nomenclature, these compounds are called 2-alkyl-4,9-naphth(2,3)imidazolediones. Since the formyl derivative could not be isolated, the parent compound was prepared by a different method. Concentrated sulfuric acid was added to a solution of 2-acetamido-3-amino-1,4-naphthoquinone in ethyl orthoformate until the color changed from bright red to an orange yellow. Vigorous bubbling followed each addition of acid. 4,9-Naphth(2,3)imidazoledione crystallized on cooling.

The screening tests on these compounds against vitamin-B_{12}-requiring *Escherichia coli* 113-3, folic-acid-requiring *Lactobacillus casei,* and purine-requiring *E. coli* B96 were quite interesting. For 50 per cent inhibition of growth of *E. coli* 113-3, where R is H, CH_3, C_2H_5, or C_3H_7, the amounts required per ml. were 0.5γ, 2γ, 5γ, and 10γ. The inhibition was partially reversed by increased vitamin B_{12} or methionine.

These compounds are also strongly inhibitory to the folic-acid-requiring *L. casei.* Those with side chains from 1 to 4 carbon atoms were equally effective, causing 50 per cent inhibition at 10γ/ml. The inhibition was only partially reversed by increase of folic acid in the medium.

With the purine-requiring *E. coli* mutant, 50 per cent inhibition was attained at 30 to 200 γ/ml. for the compounds with 1 to 4 carbon atoms in the side chain. The unsubstituted compound was the most active, 5γ/ml. The inhibition was not reversed by increase of adenine or guanine in the medium.

Work with these compounds has yielded results of little interest in the antitumor program, although Ralph Jones, University of Miami, Coral Gables, Fla., has reported that they have some activity. Thus far no compound of this type, with substituents in ring I, has been tested. We have found that direct substitution of the 4,9-naphth(2,3)imidazolediones

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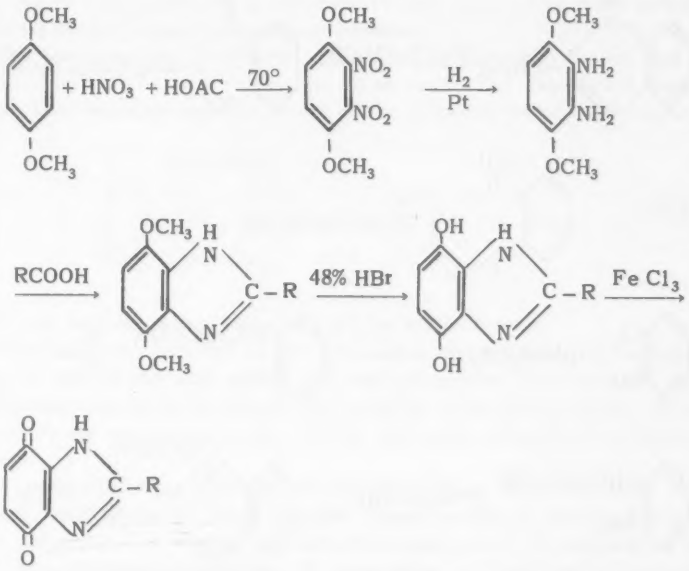
gives the 6(7)-derivative, and a series of compounds of this type is being prepared.

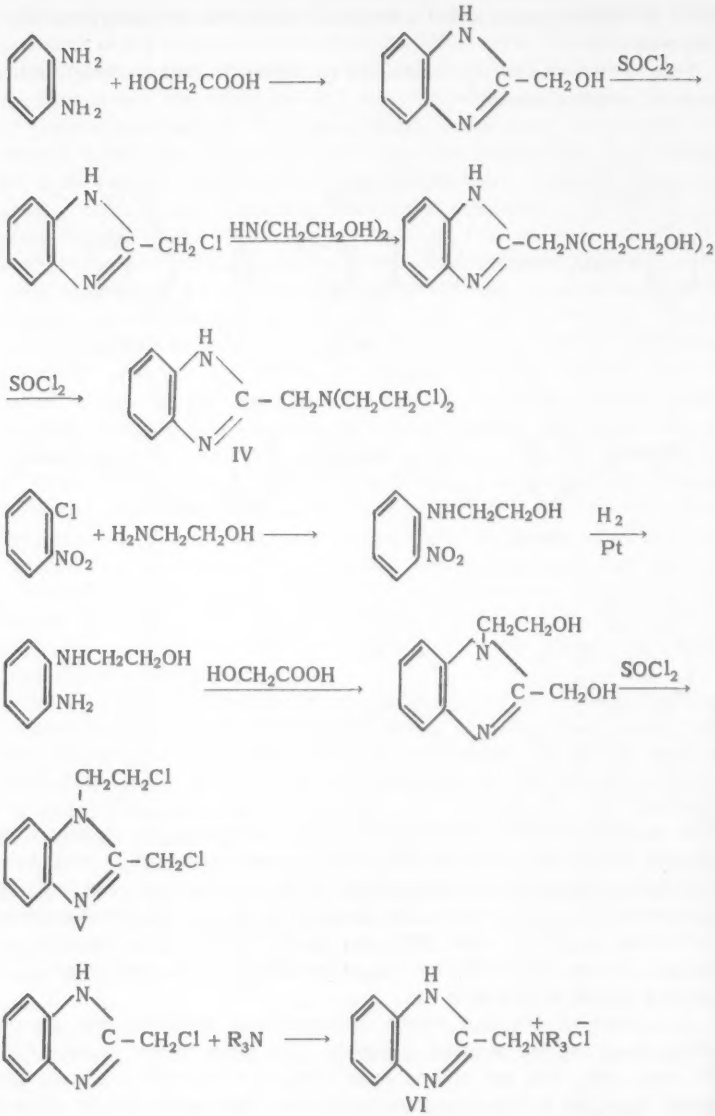
This work has now been extended to imidazole (and triazole) derivatives of *p*-benzoquinone:[2]



The nitration reaction is reported in the literature, but is poorly described. In the present work this reaction was carried out at such a rate that the temperature was maintained at 70° C. The remaining reactions proceeded normally. Due to the insolubility of the 4,7-dimethoxybenzimidazoles in 48 per cent HBr, the demethylations often proceed very slowly. These compounds have been submitted to the test program, but results are not available as yet.

A number of dimethyl ethers of substituted hydroquinones are now being used for the starting materials. The substituents include CH_3, Cl, NO_2, NH_2, SH, and others. Some of these compounds are being prepared from the 4,7-dimethoxybenzimidazole. The latter can be nitrated, sulfonated, and halogenated quite easily.

In another series of reactions, 2-chloromethylbenzimidazoles have been treated with secondary amines or tertiary amines to form either tertiary amines or quaternary compounds:

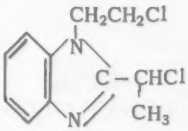


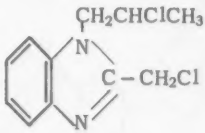
R_3N = 2-dimethylaminoethanol, 2-diethylaminoethanol, pyridine, 3-pyridine-carboxamide, 4-(2-hydroxyethyl)-5-methylthiazole, trimethylamine, and triethylamine.

Compound IV is a nitrogen mustard derivative of benzimidazole. Several derivatives of this compound have been prepared also, namely the 6-nitro, 4,7-dimethoxy, and one in which the side chain was changed to CH_3

$CHN(CH_2CH_2Cl)_2$. The preparation of additional compounds is in progress. These compounds are being tested at the present time. During the course of the present study compound IV was prepared by other workers and its activity against tumors was reported.[3]

We have prepared compound V and the corresponding 6-nitro and 6-amino derivatives, and others are being made. The chloroalkyl groups have been modified also, as shown by the following completed examples:





The test data for these compounds will be available soon.

The reactions involved in the preparation of compounds of the types IV, V, and VI are well known and need no special comment. The only difficulties encountered were in the isolation of the final products. These products are quite hygroscopic, and are not easily obtained in anhydrous form.

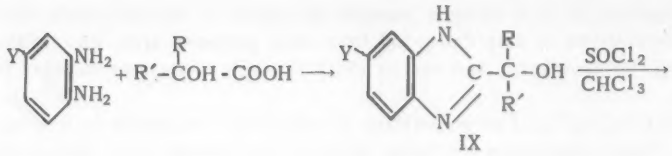
A number of benzimidazolyl analogues of folic acid have been prepared,[4] and others are being prepared. These syntheses involve 4 steps: (1) preparation of diethyl *p*-aminobenzoylglutamate; (2) preparation of 2-hydroxyalkylbenzimidazoles; (3) preparation of 2-chloroalkylbenzimidazoles; and (4) the condensation of the chloroalkyl compound with the glutamate derivative:

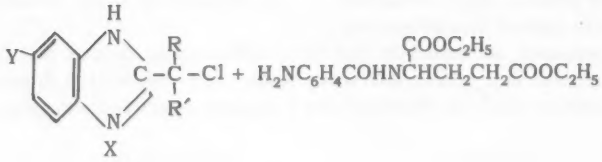
$$p\text{-}O_2NC_6H_4COCl \text{ (in benzene)} + H_2NCH(COOH)CH_2CH_2COOH \text{ (in NaOH soln.)} \xrightarrow{\text{stirring}}$$

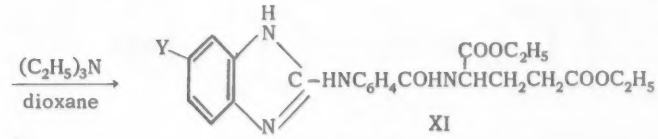
$$O_2NC_6H_4COHNCH(COOH)CH_2CH_2COOH \text{ (VII)} \xrightarrow[Pd]{H_2} H_2NC_6H_4COHNCH(COOH)CH_2CH_2COOH$$

$$\xrightarrow[HCl]{C_2H_5OH} H_2NC_6H_4COHNCH(COOC_2H_5)CH_2CH_2COOC_2H_5 \text{ (VIII)}$$

Compound VII was precipitated at a *p*H of 1 after *p*-nitrobenzoic acid was removed at a *p*H of 4 to 5. Compound VIII was precipitated at a *p*H of 8 after removing part of the ethanol under reduced pressure.







Y = H, Cl, NO_2, NH_2, CH_3. R and R′ = H or CH_3

These compounds are named as derivatives of diethyl glutamate:

Diethyl N-{4-[-1-(2-benzimidazolyl)-ethylamino]-benzoyl}-glutamate

As reported by others, we have found that N-{4-[(2-benzimidazolyl)-methylamino]-benzoyl} glutamic acid has a slight growth-promoting activity for a folic-acid-requiring *L. casei*. This is of considerable interest, since it suggests that the benzimidazole moiety acts as a formylating agent. The use of acylated imidazoles as acylating agents is being studied in our laboratories.

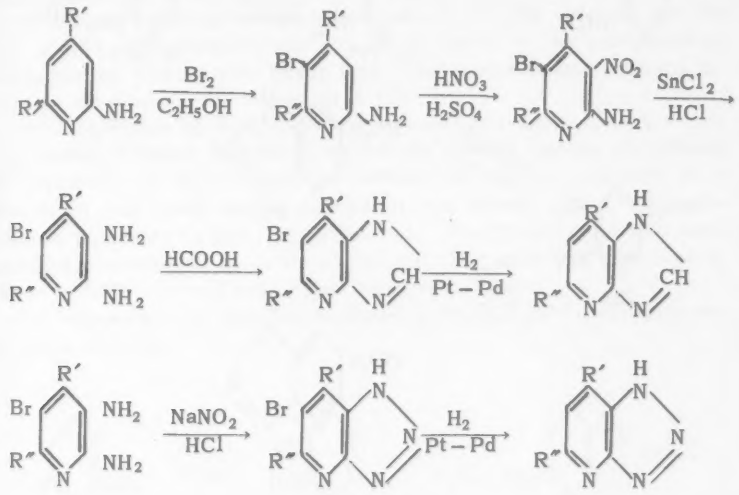
In most cases the hydroxy compounds (IX) were converted to the corresponding chloro compounds by heating with thionyl chloride in chloroform solution. The chloro compounds were usually isolated as their hydrochlorides because they were more stable than the free bases.

The yields of the final products varied from 11 to 41 per cent, which is satisfactory when one considers the nature of the work. They could be improved by further effort.

The synthesis of a number of imidazopyridines and pyrido-triazoles has been completed recently,[5] and these compounds are being tested in

our screening program at the present time. This work may be divided into 4 parts:

(1) *Preparation of methyl-substituted imidazo-[b]-pyridines and pyrido-[2,3-d]-v-triazoles:*

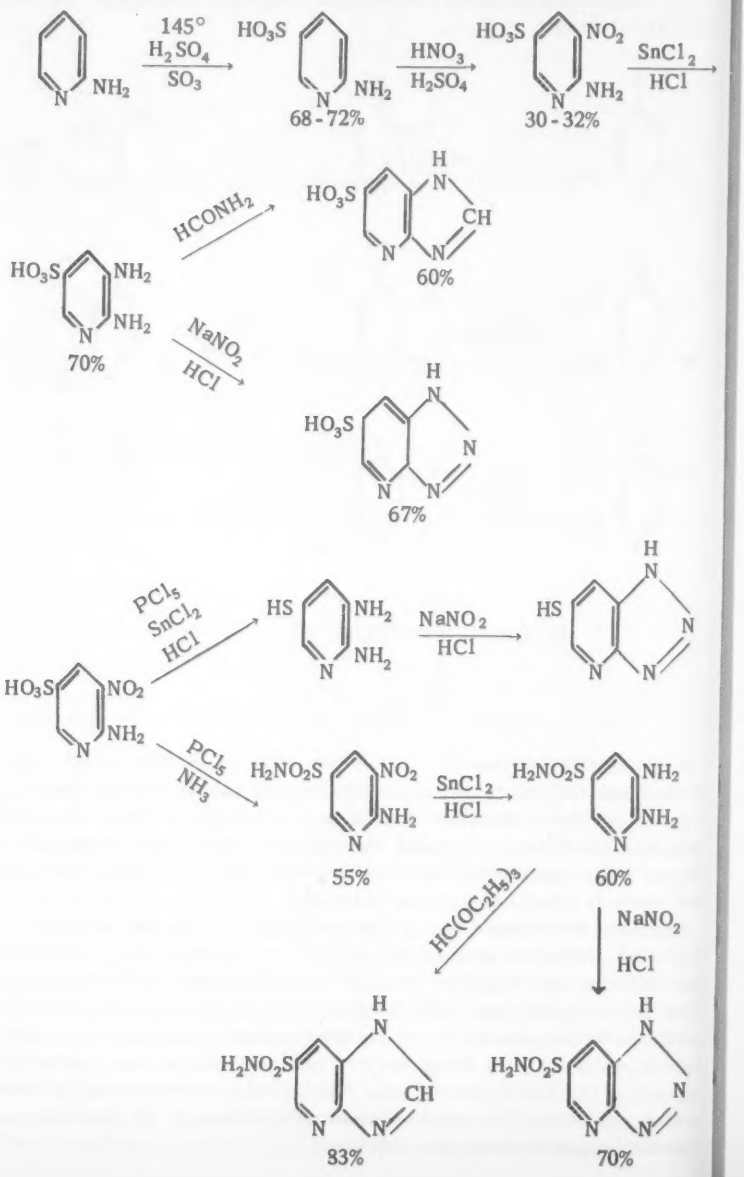


$R' = CH_3$, $R'' = CH_3$
$R' = CH_3$, $R'' = H$
$R' = H$, $R'' = CH_3$
$R' = H$, $R'' = H$

The bromination of the substituted 2-aminopyridines was carried out by the method of Case.[6] This method gave good yields of monobromo products in all cases except with 2-amino-4,6-dimethylpyridine, from which only the 3,5-dibromo compound was formed. The 5-bromo compound was obtained by acetylating the amino group and brominating the acetyl derivative in glacial acetic acid below 40°.

Nitration was carried out by the procedure of Vaughan, Krapcho, and English.[7] Reduction of the nitro group to the corresponding amino group was effected by stannous chloride in concentrated hydrochloric acid. This method gave purer products than catalytic hydrogenation. The conversions of the diamines to the corresponding imidazoles were carried out by refluxing with formic acid. The end products were isolated by removal of the excess formic acid. The triazoles were obtained by adding sodium nitrite to dilute acid solutions of the diamine. In most cases the triazole separated during the addition.

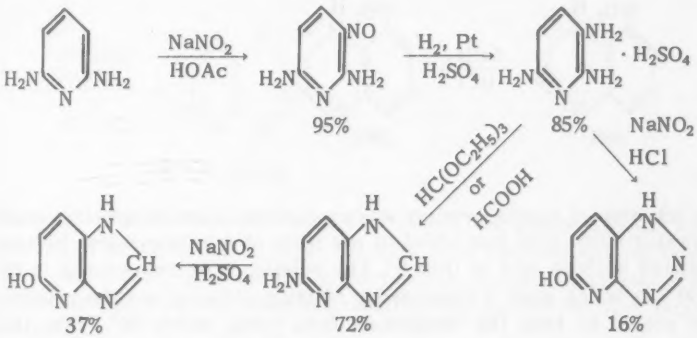
(2) *Preparation of imidazo-[b]-pyridine-6-sulfonic acid, pyrido-[2,3-d]-v-triazole-6-sulfonic acid, and related compounds:*



The sulfonation was carried out at 140 to 145° for 16 hr. with 15 to 18 per cent fuming sulfuric acid.[8] The nitration procedure was a modification of Räth's method.[9] The optimum conditions for the nitration of 2-aminopyridine-5-sulfonic acid involved the addition of fuming nitric acid to a solution in concentrated sulfuric acid at 50 to 55°. The zwitterion character of 2-aminopyridine-5-sulfonic acid probably accounts for the low yields. The diamine could not be converted to the corresponding imidazole by heating with formic acid. The imidazole was obtained by heating the diamine with formamide at 150°.

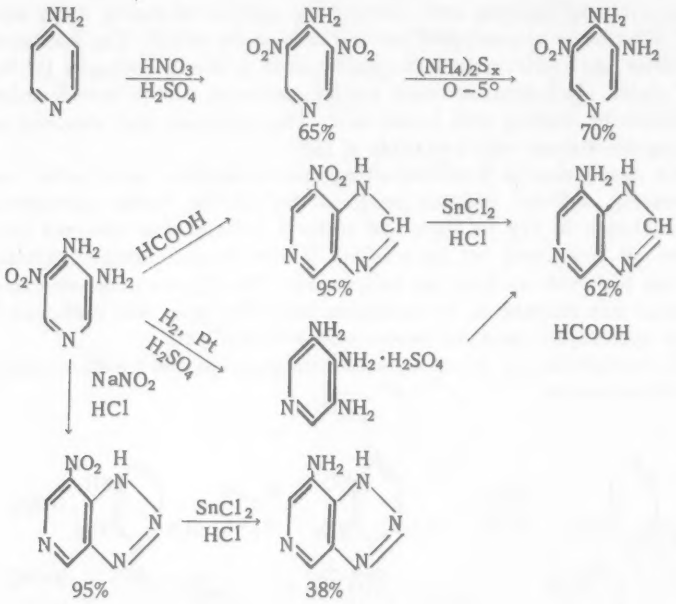
The conversion of 2-amino-3-nitropyridine-5-sulfonic acid to the corresponding sulfonyl chloride required the use of excess phosphorus pentachloride at 170 to 180°. The sulfonyl chloride was obtained as a brown oil that could not be purified. It was treated directly with ammonium hydroxide to form the sulfonamide. The 2,3-diamino-5-mercaptopyridine was isolated as its tin double salt. The latter was dissolved in dilute hydrochloric acid and treated with sodium nitrite.

(3) *Preparation of 5-hydroxyimidazo-[b]-pyridine and 5-hydroxypyrido-[2,3-d]-v-triazole:*



Attempts to prepare 2,6-diamino-3-nitropyridine by direct nitration were not very successful. A better procedure for the introduction of an amino group in the 3-position was through the 3-nitroso derivative. The nitroso compound was made by the method of Titov,[10] by adding sodium nitrite to a cold suspension of the diamine in 50 per cent acetic acid. Neutralization of the solution gave almost quantitative yields of the nitroso derivative. Hydrogenation of the latter proceeded readily, but the resulting triamine was isolated as its sulfate because of its sensitivity to air oxidation. The remaining reactions were normal in all respects and need no further comment. The low yield of the triazole was due to competing side reactions.

(4) *Preparation of 7-aminoimidazo-[c]-pyridine, 7-aminopyrido-[3,4-d]-v-triazole and related compounds:*

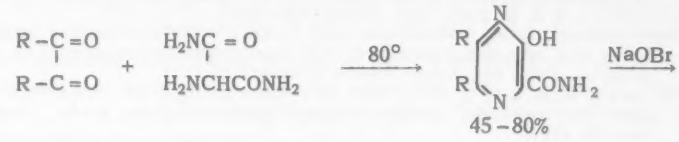


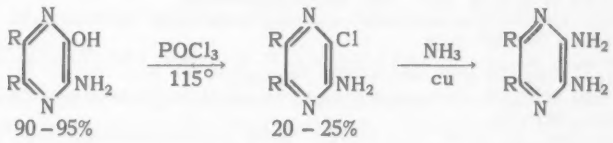
The nitration of 4-aminopyridine was carried out in two steps. One mole of fuming nitric acid was added to one mole of 4-aminopyridine in concentrated sulfuric acid at 0 to 5°. The solution was then warmed to 85 to 90°, at which point a spontaneous reaction occurred and the solution was cooled to keep the temperature from rising above 90°. When the spontaneous reaction was completed, a second mole of fuming nitric acid was added at 85 to 90°. The dinitro compound was isolated by pouring the solution onto cracked ice. The only difficulty experienced in the remaining reactions was in the removal of tin from the reduction products when stannous chloride was used as the reducing agent. The tin double salt that separated from the solution on cooling was suspended in 0.3 *N* hydrochloric acid and treated with hydrogen sulfide. Repeated treatment did not remove all of the tin. After the removal of the tin sulfide, it was found necessary to treat the end products with dilute alkali to remove the last traces of the metal.

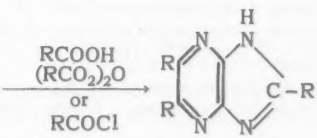
To date the test data indicate a surprisingly low antimetabolite activity for the imidazo[b]pyridines. They appear to have less activity than the benzimidazoles. The 7-nitro and 7-aminoimidazo[c]-pyridines

have shown more activity in the screening program, and they are being subjected to further tests. The pyridotriazoles have shown little or no physiological activity.

One more class of compounds deserves mention in this series, namely the imidazopyrazines. Since they are isomeric with the corresponding purines, they appeared to be of considerable interest as potential antagonists of the purines. The imidazopyrazines proved to be difficult compounds to prepare, and the synthetic methods are far from satisfactory: [11]



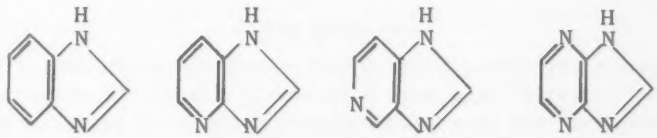




R = H or CH_3

The most difficult step was the replacement of the hydroxyl group with chlorine. The yields of 20 to 25 per cent were obtained only in those cases where 1 to 2 grams of the hydroxy amino compound was used. The yields decreased rapidly when the reaction was carried out with larger amounts.

A priori one might expect a gradual increase in anti-metabolite activity in going from benzimidazoles to imidazopyridines to imidazopyrazines:



However, as the structure becomes closer to that of the purines, exactly the opposite order is found in the examples tested to date. Only in the benzimidazole series has any appreciable anti-adenine and anti-guanine

activity and some anti-B_{12} activity been found. The activity is much weaker in the imidazopyridine series, and no activity has been found in the imidazopyrazines. This is not a final report. Other substituted imidazopyrazines are being made to determine whether or not all compounds of this series are likewise lacking in activity.

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SECTION OF GEOLOGY AND MINERALOGY

PHASE ASSEMBLAGES IN SULFIDE ORE DEPOSITS*

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Introduction

Gibbs' Phase Rule, which geologists have applied with enlightening results to the origin of igneous and metamorphic rocks, is also capable of explaining many relationships among the sulfides and associated minerals that make up metal-bearing ore bodies. The fact that the Phase Rule has not yet been widely applied to these problems is probably attributable to three hurdles:

(1) Chemical and thermodynamic data on the pertinent systems are incomplete.

(2) Sulfide ores are not always equilibrium assemblages. They are, in fact, replete with evidence of disequilibrium recorded in textures that indicate partial or complete replacement of one mineral by another.

(3) The temperature and pressure of any ore-depositing system are known only from inference and within wide limits, while the composition of the system, except for the components that are preserved as minerals, is the subject of notorious differences of opinion.

While these obstacles are formidable, they need not discourage an attempt to explore the implications of phase relationships. To compensate for the lack of chemical data, there is a wealth of description of the associations and textures of minerals in ore deposits the world over, available from studies of ores in polished section under the microscope and by other means. Textures indicating disequilibrium reflect changing conditions during or following ore deposition, and so can be highly informative in pointing the direction in which conditions are changing. Finally, while the complete composition of an ore-depositing system is unknown, it is clear that the number of solid phases – for example, minerals – that were stable together could not exceed the number that could exist in equilibrium in conformity with the Phase Rule.

The Phase Rule

Thus the Phase Rule decrees that in ores deposited over a range of temperature and pressure (2 degrees of freedom) the number of phases cannot exceed the number of components that enter into these phases. For the 3-component system Cu-Fe-S, considered in this paper, not more than 3 mineral phases can be stable together and for the 4-component

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on October 7, 1957.

system Cu-Fe-As-S not more than 4 phases can be stable. If one component, for example, sulfur, was mobile (that is, free to enter the system or be expelled from it) a third degree of freedom, activity of sulfur, is added, and the number of stable phases decreased by one.[1] In this case, 2 phases would be stable in a 3-component system and 3 phases in a 4-component system.

Basis of Diagrams

Application of these principles may be illustrated by inferred isothermal phase diagrams for a number of systems, including diagrams for several 3-component systems already presented by McKinstry and Kennedy.[2] The present paper ventures further and suggests possible stable assemblages in a 4-component system, namely, Cu-Fe-As-S.

To construct these diagrams, the plotted points representing compositions of minerals in mole per cent have been connected by joins inferred from: (1) reported co-occurrences of minerals; (2) presumed incompatibility of certain minerals that rarely or never occur together; and (3) geometrical requirements of a phase diagram.

While the joins are based on detailed evidence too voluminous to present in this abstract, the evidence for some joins is naturally more reliable than that for others (joins supported by only scanty evidence are shown in dotted lines in the accompanying illustrations, and certain areas where geometry actually demands joins have been left blank for lack of information). The diagrams are, of course, subject to confirmation or revision as thermodynamic information accumulates. In particular, since natural assemblages carry no temperature- or pressure-readings, controlled investigation very possibly will show that in some cases the pattern of joins will shift with change of temperature (or pressure); any such shifts would provide useful points on the geologic thermometer (or barometer). However, except for relationships of solid solutions and possibly for digenite, a comparison of natural assemblages suggests that the diagrams presented in this paper, although of the isothermal form, are valid for any temperature within the range of hydrothermal conditions.

Sequence of Assemblages

By studying the textures of ores from many districts and giving special attention to features such as veining and replacement of one mineral by another, various investigators have drawn conclusions as to the order in which the minerals have deposited or crystallized, and this order is sufficiently uniform to suggest a "usual" sequence or "general mineral succession"[3] for which various explanations have been offered. McKinstry and Kennedy[2] have suggested that sequence becomes consistent with chemical relations if it is regarded, not as a sequence of individual minerals, but as a sequence of phase assemblages. In particular, for a

constant ratio among the metallic elements, a sequence of assemblages, each richer in sulfur than the preceding one, would call for the disappearance of one mineral and the appearance of another as the aggregate composition of the solid phases progressed toward the sulfur corner (A-B, FIGURE 1), and this series of appearances and disappearances

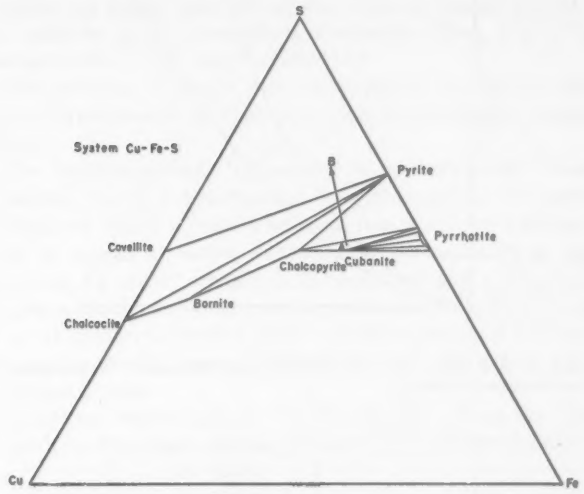

FIGURE 1. Inferred phase diagram for the system Cu-Fe-S. (After McKinstry and Kennedy.[2]) Reproduced by permission of *Economic Geology*.

corresponds closely with the "usual" sequence. Similar relations apply to other "volatiles" such as arsenic and antimony.

As an explanation for this relationship, which might almost be called the "law of increasing sulfur," Kennedy pointed to typical equilibrium curves (FIGURE 2) for reactions involving a volatile phase, and showed that phases richer in sulfur are favored by: (1) increase in partial pressure of S at constant temperature; (2) decrease in temperature at constant partial pressure; or (3) combinations of 1 and 2 (FIGURE 3).

The Tetrahedral Model Cu-Fe-As-S

A 4-component system can be depicted by using a model such as the tetrahedron shown in FIGURE 4, whose faces, in this instance, represent 4 ternary systems:

Cu-Fe-S
Cu-As-S
Fe-As-S
Cu-Fe-As

The last of these 4 contains no mineral phases additional to those that

appear on the edges of the other triangular faces. The other 3 systems will be discussed in order.

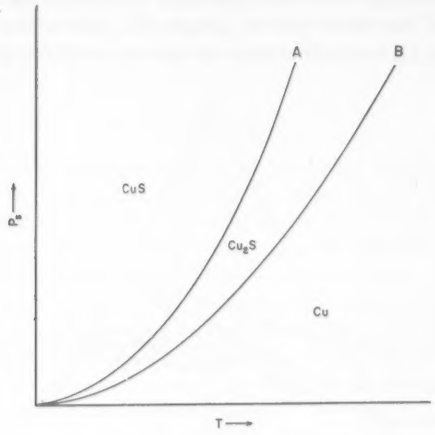

FIGURE 2. See text. (After McKinstry and Kennedy.[2]) Reproduced by permission of *Economic Geology*.

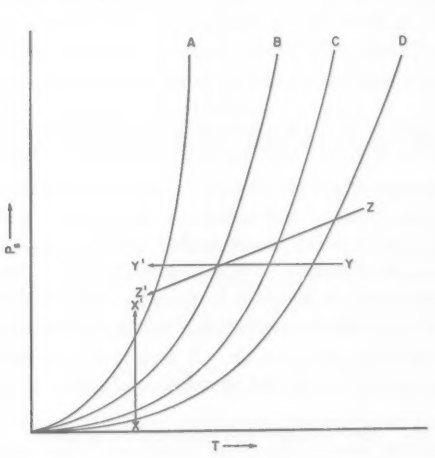

FIGURE 3. See text. (After McKinstry and Kennedy.[2]) Reproduced by permission of *Economic Geology*.

Ternary System Cu-Fe-S

The diagram postulated in FIGURE 1 is incomplete in that it does not show the fields occupied by certain solid solutions that are stable in

the higher temperature ranges but break down on cooling, as the limits of these solid solutions are in most cases either undetermined or subject to conflicting evidence. Such fields would adjoin or straddle the compositions of chalcopyrrhotite,[4,5] valleriite, chalcopyrite, bornite, and digenite (Cu_9S_5).[6,7] Thus, although the diagram is oversimplified, it brings out certain interesting relationships:

(1) Pyrite is stable with all of the common mineral phases in the system, with the possible exception of cubanite. Thus, it is a "persistent" mineral with a wide range of stability.

(2) The stability of pyrite with chalcopyrite, bornite, or chalcocite precludes coexistence of covellite with pyrrhotite, cubanite, chalcopyrite, or bornite.

(3) The long-recognized "antipathy" of pyrrhotite and bornite[8] can be accounted for by a hypothetical join, chalcopyrite-Fe (native), an association not found in nature because iron would be oxidized in the presence of oxygen or water. However, the compatibility of magnetite (Fe_3O_4) with Fe under metallurgical conditions and with chalcopyrite in many ore occurrences would indicate a join-plane chalcopyrite-magnetite-Fe in the system Cu-Fe-O-S which would appear as a join chalcopyrite-Fe on the ternary plane Cu-Fe-S and preclude a join connecting pyrrhotite and bornite.

(4) Increasing sulfur content, as, for example, along the line A-B, would result in a sequence of assemblages in the following order:

cn-po-cp*
po-cp-py
cp-py-bn
py-bn-cc
py-cc-cv

This order, except for reversal of pyrite and pyrrhotite, calls for a disappearance of one mineral and appearance of another in order identical with the reported "usual" sequence:

py, po, cp, bn, cc, cv

Ternary System Cu-As-S

The minerals on the Cu-As edge of this triangle (FIGURE 5) are arsenical copper (γ phase)[9] and a series of copper arsenide minerals found chiefly in the Keweenaw district of Michigan. A triangle Cu-domeykite-chalcocite in the low-sulfur corner of this diagram would embrace the copper-mineral assemblage of this district.

In the ternary system the most important minerals are tennantite and enargite (which has an isomorph, luzonite). Occurrences, notably at Butte, Mont., indicate a join chalcocite-enargite rather than tennantite-covellite.

*For abbreviations of mineral names in the text and in the diagrams see ADDENDUM to this paper.

On the As-S edge of the triangle are orpiment and realgar, which rarely occur with the copper minerals. The dotted joins are inferred from experimental work by Gaudin and Dicke.[10]

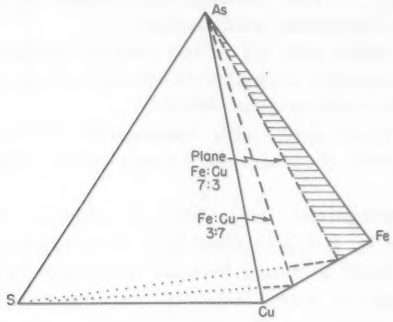

FIGURE 4. Tetrahedral model for the four-component system Cu-Fe-As-S showing positions of the planes for FIGURES 8 and 9.

FIGURE 5. Inferred joins in the system Cu-As-S.

Ternary System Fe-As-S

FIGURE 6 shows the iron-arsenic-sulfur face of the tetrahedron. Its most striking feature is that löllingite, arsenopyrite, and pyrite all lie on a straight line, so that löllingite and pyrite are incompatible; as a matter of observation, they never occur together. Another pair not observed in contact with each other is pyrrhotite-löllingite.[11] This may be due to a join arsenopyrite-Fe, analogous to the suggested join chalcopyrite-Fe. Realgar and orpiment on the As side occur as rarely with the

iron minerals as with the copper minerals. At Getchell, Nev.,[12] orpiment apparently is contemporaneous with pyrite, while realgar is later and contemporaneous with marcasite.

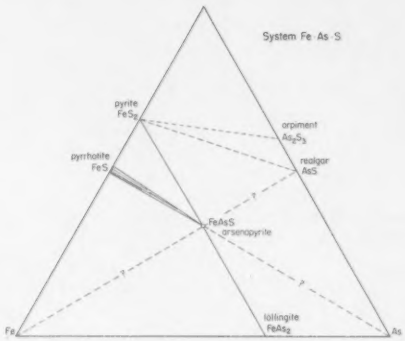

FIGURE 6. Inferred joins in the system Fe-As-S. (A join pyrite-arsenic is alternative to the join arsenopyrite-realgar and may be more probable.)

The Quaternary System Cu-Fe-As-S

Three of the faces of the 4-component tetrahedron are shown together in FIGURE 7. From these the tetrahedron can be visualized by mentally folding up the 2 outer sub-triangles so that the 2 apices labeled "As" meet at a point above the base. As this diagram is not convenient for showing joins that pass from one face to another through the interior of the tetrahedron, 2 sections have been cut through the tetrahedron, as indicated in FIGURE 4, representing high and low ratios of iron to copper. FIGURE 8 is intended primarily to permit a comparison of the two sections; part of the section for a ratio of Fe to Cu of 3:7 is shown on a larger and more accurate scale in FIGURE 9.

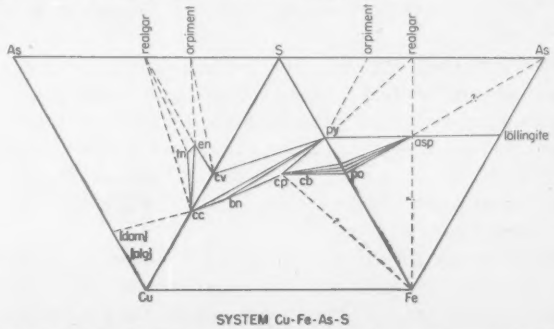

FIGURE 7. Three of the ternary systems in the quaternary system Cu-Fe-As-S.

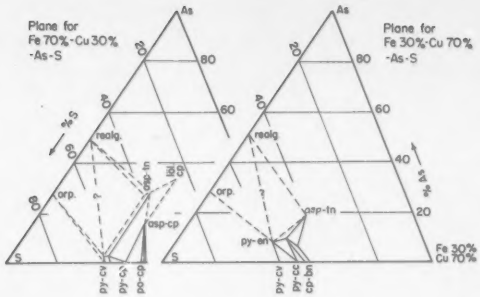

FIGURE 8. Two sections through the quaternary system Cu-Fe-As-S along planes shown in FIGURE 4.

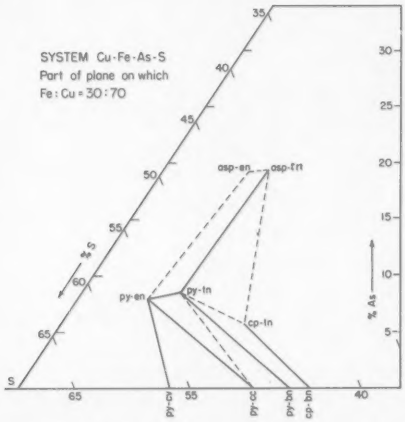

FIGURE 9. Part of one of the planes shown in FIGURE 8. Lines connect piercing-points of two-phase joins and represent three-phase assemblages.

On these sectional diagrams, the lettered points are the piercing-points at which any join-line connecting 2 phases in space passes through the section. Accordingly the lines on the section are the traces of what in 3 dimensions are triangular planes joining any 3 phases that are presumed to be capable of existing in equilibrium together. Thus, the lines represent 3-phase, and the quadrilaterals (or in some cases triangles), 4-phase assemblages.

Comparison of the 2 sections in FIGURE 8 shows: (1) pyrrhotite and probably löllingite, present in the high-iron section, are absent in the high-copper section; and (2) the copper sulfide and copper-iron sulfide minerals are stable in assemblages much higher in arsenic where the copper content is high than when it is low.

A portion of the higher-copper section shown in FIGURE 9 illustrates, among other things, the sequence at Butte, Mont., thoroughly studied by Sales and Meyer.[13] There the assemblage chalcopyrite-bornite-tennantite is early and, on the deeper levels of the copper zone, is replaced by pyrite-enargite-chalcocite and ultimately by pyrite-enargite-covellite. This sequence of assemblages would follow the course of a line drawn from some point on the cp-bn-tn join (at the right) toward the sulfur corner or a low point on the S-As edge. It shows graphically why tennantite gives way to enargite at about the stage at which bornite and chalcopyrite are succeeded by pyrite and chalcocite.

Districts as Mega- Systems

Although the systems considered thus far are of the dimensions of a polished section or, at most, of a subzone in a vein system, the phase rule imposes no limit on the size of a system. In fact, a whole mine or mining district or, for that matter, a metallogenetic province can be considered as a system, although such a large system is, of course, never uniform or in equilibrium throughout. Nevertheless, certain districts have their characteristic mineral assemblages, and it is rare in any district to find all of the minerals that *could* form from the elements available. Thus, in Central Peru (including Cerro de Pasco and Morococha), bornite and primary chalcocite are rare and löllingite is rarer, if it occurs at all. The iron-copper minerals are, with few exceptions, those whose compositions lie within the portion of the 4-component model bounded by the corners arsenopyrite, pyrrhotite, chalcopyrite, pyrite, tennantite, and enargite, together with antimonial analogues.

In FIGURES 10 and 11 the position of the name of each of several districts is intended to indicate roughly the characteristic proportion of iron to copper for the district although, lacking quantitative analytical data, this can be gauged only by the mineral assemblages observed and is not intended to be precise. The mineral assemblages present are indicated by the heavy line opposite the name of the district.

FIGURE 10 is the pertinent part of the ternary diagram Cu-Fe-S. FIGURE 11 is based on a section through the quaternary diagram Cu-Fe-As-S on which the arsenic content is 3 mole per cent. For other percentages of arsenic, some of which might be more realistic for certain of the districts, the positions of piercing points, and consequently, the geometrical shapes of fields, would be different, but the phase assemblages would be the same until the As content reaches that of enargite (12.5 per cent). In some districts mineralization started with the low-sulfur assemblage pyrrhotite-chalcopyrite, and continued to the later assemblage pyrite-enargite. In others, as in most of the porphyry copper deposits in the Southwest, only the assemblage pyrite-chalcopyrite with or without bornite is represented in any quantity.

Other Components and Systems

The actual sulfide assemblages in copper-bearing districts include greater or lesser amounts of the sulfides of other elements of which zinc and lead are usually the most abundant. In general, though there are exceptions, an added metallic element merely adds one to the dimensions of a simple phase diagram without adding to the number of iron-sulfide or copper-sulfide bearing phases.

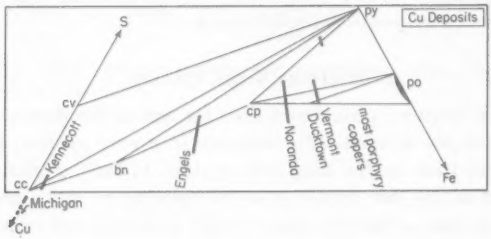

FIGURE 10. Mineral assemblages in several mining districts plotted on a portion of the ternary diagram Cu-Fe-S. Districts are: Michigan (Keweenaw Peninsula); Kennecott, Alaska; Engels, Calif.; Noranda, Quebec; Ducktown, Tenn.; Vermont Copper Belt; most (southwest United States) porphyry copper deposits.

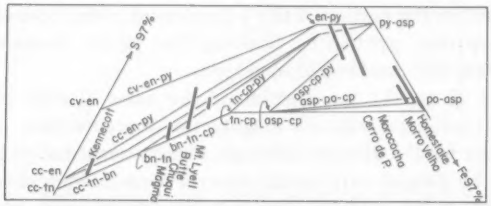

FIGURE 11. Some mining districts in which arsenical minerals are present, plotted on a section through the quaternary system Cu-Fe-As-S at composition As, 3 per cent. Corners (beyond margins of the figure): Cu, Fe, and S each 97 per cent (atomic proportions). Kennecott, Alaska; Magma, Ariz.; Chuquicamata, Chile; Butte, Mont.; Mt. Lyell, Tasmania; Morococha, Peru; Cerro de Pasco, Peru; Morro Velho, Brazil (gold); Homestake, S. Dak. (gold).

Zinc occurs in sulfide ores only as sphalerite (ZnS), or its dimorph, wurtzite, and forms no arsenical (or antimonial) compounds. Except for the extent to which sphalerite takes iron or copper into solid solution, zinc does not combine with these elements to form complex sulfide minerals.

Lead occurs chiefly in the form of galena and, like zinc, does not combine with copper or iron to form sulfides. It does form an extended

series of arsenic and antimony sulfides for which phase diagrams are presented in the recent paper.[2]

Although the system Cu-Fe-As-S is used for illustration in the present paper, antimony, a common companion to arsenic, adds complexity to the system chiefly on account of the two isomorphous series, tetrahedrite-tennantite and famatinite-luzonite. In general, however, the Cu-Fe-Sb-S system is so similar to the corresponding arsenical system that the five-component system could be represented by substituting AsSb for As in the corner of the four-component tetrahedron without greatly changing the configuration of the model.

Acknowledgments

Robert M. Garrels, who read the present manuscript, has made a number of helpful suggestions. The paper has benefited from the work of several graduate students at Harvard University, notably from a course report by Ulrich Peterson on the Cu-Fe-S system.

ADDENDUM

Notes. In the diagrams accompanying this paper, mineral names are abbreviated in accordance with the list recommended by Chace.[14] This list, based on usage that had already received considerable acceptance, is carefully planned to avoid duplication, and is the nearest existing approach to a set of standard symbols (note that, except for native elements, the abbreviations are not capitalized).

Abbreviations for minerals in the system under discussion are:

alg	algodonite	lo	löllingite
As	arsenic	lz	luzonite
bn	bornite	ms	marcasite
cc	chalcocite	orp	orpiment
Cu	copper	py	pyrite
cn	cubanite	po	pyrrhotite
cp	chalcopyrite	rl	realgar
dom	domeykite	S	sulfur
en	enargite	tn	tennantite
Fe	iron	wurt	wurtzite

Since preparing this abstract, my attention has been called to a recent work[15] that presents diagrams somewhat similar to those in the present paper. Unfortunately, the text is in Russian, and I have not yet received a translation.

While the present article was in press, the following papers relating to the same topic were presented at the meeting of the Geological Society of America held in Atlantic City, N. J., November 4, 5, and 6 and are abstracted in the program of the 1957 Annual Meeting: Thermochemical Data, Mineral Associations and the Lindgren Classification of Ore Deposits, by H. D. Holland; Upper Stability Curve of Covellite, by G. Kullerud; and Phase Relations in the System $CoAs_2$-$NiAs_2$-$FeAs_2$-As, by E. H. Roseboom, Jr.

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SECTION OF BIOLOGY

THE ROLE OF VITAMIN E AT THE ENZYMATIC LEVEL*

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Introduction

Our current knowledge of vitamin E, or α-, β-, γ-, δ-, ϵ-, ζ-, and η-tocopherol (the various mono-, di- and trimethyl-tocols), stems from its discovery more than thirty years ago as a fat-soluble factor necessary for reproduction in the rat. The chemical nature and synthesis of tocopherol have since been established, and the vitamin has been shown to be widely distributed among plants and animals. Detailed studies have been made of the effects of vitamin E deficiency and addition *in vivo* and *in vitro* with various tissues and cell-free systems, and the vitamin has been associated with a wide variety of biological processes.[1-4] In addition to its role as an antisterility factor for the laboratory rat, tocopherol has been reported to be necessary for the structural and functional maintenance of skeletal, cardiac, and smooth muscle, and the peripheral vascular system in a number of animals. The muscular dystrophy associated with vitamin E deficiency is accompanied by a small and at times inconsistent increase in oxygen consumption of the dystrophic tissue and by alterations in chemical composition and functional behavior. Many of these effects have been attributed to the action of tocopherol as an intracellular antioxidant, namely, its protective action in inhibiting the oxidation of unsaturated fats and other oxygen-sensitive substances, such as vitamins A and C, during storage. It is generally believed, however, that the nonspecific action of tocopherol as a physiological antioxidant represents a secondary role. A number of recent reviews[1-4] have emphasized that its primary mechanism of action in the cell is unknown, although some believe that the vitamin acts at the enzymatic level.

Houchin and Mattill,[5,6] who reported an increased rate of succinic acid oxidation by hamster dystrophic muscle, postulated the role of tocopherol as an inhibitor of cytochrome c reduction. Basinski and Hummel[7] reported, however, that under comparable conditions the succinic dehydrogenase system is unaffected. Although general disturbances in

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on October 14, 1957. It constitutes Contribution No. 196 of the McCollum-Pratt Institute. The investigation it describes was supported in part by a research grant from the Muscular Dystrophy Associations of America, Inc., New York, N. Y., and by Research Grant No. 2332 from the National Institutes of Health, Public Health Service, Bethesda, Md.

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phosphorylation mechanisms have been noted in dystrophic muscle, including diminution of the creatine[6,8] and adenosine triphosphatase [9] levels, no decrease has been observed in oxidative phosphorylation in preparations of heart tissues of dystrophic rabbits.[10] More recent reports, however, indicate that there is a lowering of the P:O ratio of vitamin E deficient preparations (see page 46).

In most studies of the effects of vitamin E and its derivatives on isolated enzyme systems *d*-α-tocopheryl phosphate was used because of its greater solubility in water and its implied involvement in energy transformations by virtue of its phosphate group. The phosphate ester, however, markedly inhibited most enzymes, including succinic oxidase,[11] diphosphopyridine nucleotidase,[12] trypsin,[13] liver acid phosphatase,[14] hyaluronidase,[15] liver esterase,[16] and lipoxidase.[17,18] Rabinovitz and Boyer[19] have ascribed the inhibitory effects of α-tocopheryl phosphate to its properties as an anion with a large nonpolar group.

Chemical studies demonstrating reversible oxidation products of tocopherol suggest that vitamin E may have its primary role as a carrier in biological oxidation-reduction reactions, perhaps as a cofactor or part of a cofactor molecule, of an enzyme system. Boyer[20] isolated and tentatively characterized as an expoxide an intermediary biologically active, reversible oxidation product of *d*-α-tocopherol prepared by ferric chloride oxidation in the presence of 2,2′-dipyridyl. The product, designated as *d*-α-tocopheroxide, is readily reduced to *d*-α-tocopherol by ascorbic acid or converted to *d*-α-tocopherylquinone by exposure to dilute acid. Martius and Eilingsfeld[21] have recently reported that tocopheroxide is not an epoxide, but rather an acetal of α-tocopherylquinone. It is possible that the tocopherol free radical of Michaelis and Wollman[22] is an intermediate in the reversible transformation between tocopherol and tocopheroxide. The *d*-α-tocopherylquinone can be reduced to the corresponding hydroquinone by ascorbic acid or converted to tocopherol by means of an HCl-ascorbic acid reduction procedure recently described by Harrison *et al.*[23]

In the present work, which has been carried out during the last three years, the possibility suggested itself that, if tocopherol is involved in biological oxidation reductions, it might conceivably act as a carrier in the oxidation of reduced pyridine nucleotides. Evidence has now been obtained to demonstrate that mammalian skeletal and heart muscle DPNH*-and succinate-cytochrome c reductases inactivated by aging or fat-solvent-extraction procedures are specifically and fully restored by tocopherol. The added tocopherol acts in a catalytic fashion in the sequence of electron transport just before cytochrome c, and there is a competitive inhibition of antimycin A with tocopherol. In further support

*The following abbreviations are used throughout the text: DPN and DPNH, oxidized and reduced diphosphopyridine nucleotide, respectively; TPN and TPNH, oxidized and reduced triphosphopyridine nucleotide, respectively.

of a function for vitamin E as a cofactor of cytochrome c reductase are data showing that inactivation of the enzyme under given conditions occurs with a concomitant dissociation from the enzyme of vitamin E. The latter is present in amounts commensurate with that of other established cofactors. Detailed reports of most of these studies have already appeared.[24-31]

Effect of Tocopherol on DPNH Oxidation

In order to test the possibility that vitamin E is involved in the biological oxidation of reduced pyridine nucleotides, the effect of *d*-α-tocopherol on the enzymatic oxidation of DPNH in the presence of various tissue extracts including that of animals, plants, and microorganisms was examined. The problem of suspending the fat-soluble vitamin E in aqueous phase was overcome by homogenizing an alcohol solution of *d*-α-tocopherol with a buffered phosphate solution of crystalline bovine serum albumin in a Tenbroeck glass homogenizer to yield a stable aqueous suspension of tocopherol (ranging to approximately 5 mg./ml.) in a 15 per cent ethanol – 0.2 per cent albumin solution. Cell-free preparations of rat skeletal muscle showed the most consistent and marked increases in the rate of DPNH oxidation upon addition of tocopherol, the increased rates ranging from 2- to 6-fold depending upon the preparation used (FIGURE 1). The addition of cytochrome c to the reaction mixture resulted in a twofold increase in both DPNH oxidase activity and the tocopherol enhancement effect. That tocopherol acted at a point between DPNH and the cytochrome c level was shown by its stimulation of cytochrome c reductase, but not of cytochrome oxidase.

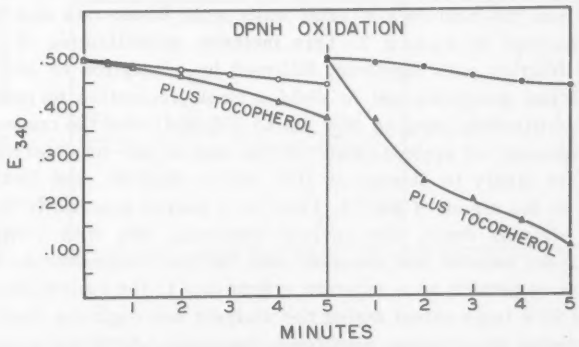

FIGURE 1. Enhancement effect of added tocopherol on DPNH oxidase. Two different preparations of partially purified rat skeletal muscle particulate DPNH oxidase (approximately 100 μg. protein) were assayed[26] with and without added *d*-α-tocopherol suspended in ethanol-albumin. The control reaction mixtures included the same concentration of ethanol-albumin, but without tocopherol.

Extraction of Enzyme by Organic Solvents

In order to demonstrate the tocopherol enhancement effect with each new enzyme preparation, it was necessary to store the enzyme fraction at -15° for 2 to 10 days with occasional thawing. The assumption that the enzyme system is accompanied by a lipide component, possibly related to tocopherol, which dissociates from the protein under the above conditions prompted an attempt to remove the presumed "tocopherol-like cofactor" by extraction with various nonpolar solvents. The enzyme preparation (1 to 5 ml.) was extracted by shaking with an equal volume of cold organic solvent for approximately 1 to 2 min. in a test tube, followed by centrifugation at about 500 × *g* for 30 sec. The nonpolar solvent was conveniently removed by means of a pipette with a rubber pressure bulb. Successive extractions were performed in the same manner with fresh portions of solvent. Of 24 solvents tried, isooctane (2,2,4-trimethylpentane) proved to be the most efficient in that it resulted in the largest decrease in enzyme activity (to one-tenth or one-twentieth of the original value), which could then be completely restored upon addition of *d*-α-tocopherol to the reaction mixture. Cyclohexane, Skellysolve, petroleum ether, and hexane were less effective, while other solvents, for example, chloroform, benzene, and carbon tetrachloride, caused an irreversible inactivation of the enzyme.

Purification and Characterization of Cytochrome c Reductase

Before proceeding with a detailed study of the effects of isooctane extraction on the DPN-cytochrome c reductase, the rat skeletal muscle enzyme from the hind legs of adult white male Wistar rats was purified, as summarized in TABLE 1. This included solubilization of the particulate fraction with digitonin, followed by adsorption to and elution from calcium phosphate gel to yield a final preparation representing a 60-fold purification (ranging from 40- to 150-fold) over the crude extract, with a recovery of approximately 10 per cent of the total activity. The enzyme is stable to storage at -15° and to dialysis, and has optimal activity in the region of *p*H 7.5. There is a marked specificity for DPNH as the electron donor, the maximal enzymatic rate with TPNH being about 20 per cent of that observed with DPNH. Succinate-cytochrome c reductase is present as a separate system and in the earlier fractions is removed to a large extent during the dialysis and digitonin fractionation steps. Under given aging conditions succinate-cytochrome c reductase can be obtained relatively free of the DPN enzyme.

Although examination with the Beckman DU spectrophotometer failed to detect flavin and cytochrome components in the concentrated particulate enzyme (Fraction III), the sensitive split beam recording spectro-

TABLE 1

PURIFICATION OF DPN-CYTOCHROME c REDUCTASE FROM RAT SKELETAL MUSCLE[26]

Fraction No.		*Total units**	*Total protein mg.*	*Specific activity*	*Percentage recovery*
I	Crude extract	228,000	820	277	100
II	Dialyzed supernatant solution	134,000	294	455	59
III	Resuspended high speed pellet	84,000	24	3,500	37
IV	Digitonin-treated	48,000	17	2,820	21
	1st Ca_3 (PO_4) gel eluate	5,040	3.8	1,330	
	2nd Ca_3 (PO_4) gel eluate	1,920	2.1	914	
	3rd Ca_3 (PO_4) gel eluate	17,400	1.15	15,100	
	4th Ca_3 (PO_4) gel eluate	4,050	0.25	16,200	
	5th Ca_3 (PO_4) gel eluate	3,900	0.12	32,500	
V	Combined 3rd, 4th, 5th gel eluates	25,350	1.52	16,700	11

* $\Delta E_{550\ m\mu} \times 10^3$ per 2 min.

photometer* showed the presence of a number of heme components. The steady state oxidized difference spectrum (FIGURE 2a) of the partially purified particulate enzyme with DPNH as the electron donor reveals the presence of cytochrome $c + c_1$, *b*, and *a* (+ cytochrome a_3). This was confirmed by the even more sensitive low temperature "apparent absolute absorption spectrum"[32] with sodium dithionite as the reducing agent (FIGURE 2b). The presence of cytochromes *b* and *c* in the particulate

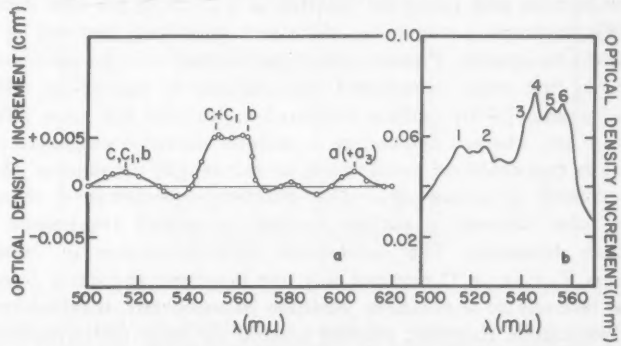

FIGURE 2. (a) Steady state oxidized difference spectrum of the rat skeletal muscle particulate DPNH oxidase system.[27] (b) Low temperature "apparent absolute absorption spectrum" of the rat skeletal muscle particulate DPNH oxidase system.[27]

*Performed by Britton Chance and Ronald W. Estabrook at the Johnson Research Foundation of the University of Pennsylvania, Philadelphia, Pa.

preparation does not mean that these components are necessarily on the main pathway of electron transfer between DPNH and oxygen. Detailed kinetic studies may clarify this point. No flavin requirement by the enzyme has been demonstrated even after precipitation of the protein with ammonium sulfate at acid *p*H as well as prolonged dialysis.

At a final concentration of 5×10^{-4} M, Fe^{++}, Cu^{++}, Zn^{++}, and Co^{++} caused 50 to 100 per cent inhibition, while Mn^{++}, Ca^{++}, $Bo_3^{=}$, $MoO_4^{=}$, Fe^{++}, and Mg^{++} were without effect. The metal binding agents α-, α-dipyridyl and 8-hydroxyquinoline, but not azide, at 5×10^{-3} M were inhibitory to the extent of 40 to 50 per cent. The sulfhydryl poison p-chloromercuribenzoate at concentrations of 1×10^{-4} and 5×10^{-4} M caused 60 to 100 per cent inhibition, which was not reversed by addition of glutathione or cysteine. The cytochrome c reductase at all stages of purity is markedly inhibited by extremely low concentrations of antimycin A. Final concentrations of antimycin A as low as 0.002 γ per ml. caused up to 90 per cent inhibition, suggesting the presence of a factor between DPN and cytochrome c heretofore not reported for a soluble system.

The succinate-cytochrome c reductase of rat skeletal muscle and the DPN- and succinate-cytochrome c reductases of bovine heart have been purified in essentially the same manner and exhibit the same over-all properties, including marked sensitivity to antimycin A, as described for the rat DPN enzyme.

Isooctane Extraction of Enzyme

FIGURE 3 demonstrates that 3 to 5 extractions of the particulate rat enzyme fraction with isooctane resulted in a 75 to 95 per cent decrease in DPN-cytochrome c reductase, which was completely restored by addition of *d*-α-tocopherol. Further extraction resulted in a progressive loss of activity that could be reversed only partially by subsequent addition of the vitamin. DPNH oxidase exhibited essentially the same behavior (FIGURE 4a), whereas cytochrome c oxidase showed a progressive loss of activity that could not consistently be restored by subsequent addition of tocopherol (FIGURE 4b). The succinate-cytochrome c reductase system also showed a similar striking tocopherol requirement after isooctane treatment. The solubilized DPN-cytochrome c reductase (Fraction V, TABLE 1) required only one isooctane extraction to reduce enzyme activity to a minimum. Addition of tocopherol resulted in complete restoration. However, purified soluble pig heart DPN-cytochrome c reductase and pig liver TPN-cytochrome c reductase* showed no tocopherol effect before or after isooctane extraction.[33,34] The isooctane extraction procedure has also made it possible to demonstrate a *d*-α-tocopherol stimulation of cytochrome c reductase in cell-free prepara-

*Provided by H. R. Mahler and B. L. Horecker, respectively.

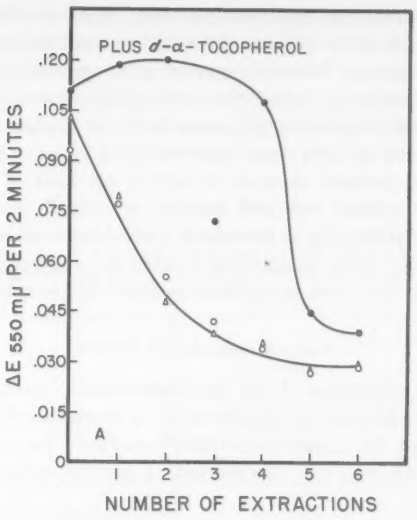

FIGURE 3. Effect of increasing number of isooctane extractions and subsequent reactivation by tocopherol on DPN-cytochrome c reductase of a rat skeletal muscle particulate preparation.[27]

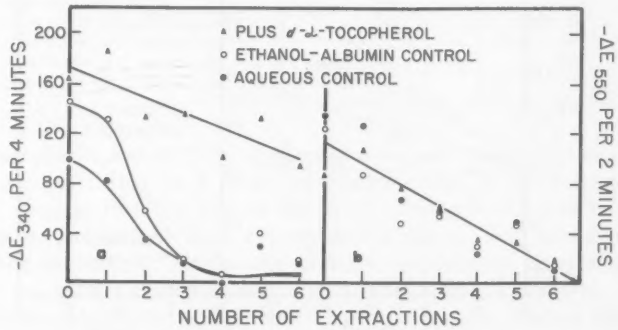

FIGURE 4. (a) Effect of increasing number of isooctane extractions and subsequent reactivation of tocopherol on DPNH oxidase of a rat skeletal muscle particulate preparation.[27] (b) Effect of increasing number of isooctane extractions and subsequent addition of tocopherol on cytochrome c oxidase of a rat skeletal muscle particulate preparation.[27]

tions of rat liver, yeast, *Neurospora*, soybean leaves, hamster skeletal muscle, and human pectoral muscle.[27]

The concentration of lipide on a dry weight basis in the rat particulate

preparations varies from 60 to 65 per cent. Three to five extractions of the enzyme resulted in only a 10 to 15 per cent decrease in the total lipide of the fraction, whereas enzyme activity was lowered by almost 90 per cent. Similarly, isooctane extraction of a purified beef heart succinic oxidase preparation provided by D. E. Green resulted in a 90 per cent decrease in DPN- and succinic-cytochrome c reductase activities with a concomitant removal of only 5 per cent of the lipide. The addition of tocopherol restored enzyme activity.[27] As described in a later section, cytochrome c reductase can also be decreased almost to zero under given aging conditions without employing isooctane extraction, and the activity then completely restored by tocopherol.

Site of Tocopherol Action

The striking inhibition of the digitonin-treated, unextracted rat DPN-cytochrome c reductase by antimycin A is competitive with tocopherol, as demonstrated by Lineweaver-Burk [35] analysis (FIGURE 5); in fact, antimycin A inhibition can be reversed in part by subsequent addition of tocopherol.

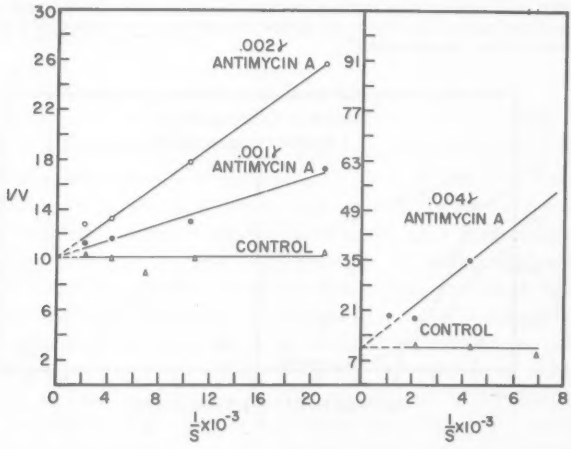

FIGURE 5. Competitive inhibition of antimycin A with tocopherol in the DPN-cytochrome c reductase system as demonstrated by the Lineweaver-Burk plot.[35] The DPN-cytochrome c reductase assay was performed in the described manner,[26] using digitonin-solubilized rat skeletal muscle enzyme (500 μg. protein per 3.0 ml. reaction mixture) and *d*-α-tocopherol (1.4×10^{-4} M final concentration) suspended in 15 per cent ethanol-2 per cent bovine serum albumin. The indicated quantities of antimycin A added are expressed in final concentrations per milliliter of reaction mixture.

When 2,6-dichloroindophenol was used as the electron acceptor in place of added cytochrome c, there was no inhibition by antimycin A of dye reduction. Extraction with isooctane caused no significant change without or with subsequently added tocopherol, whereas the rate of cytochrome c reduction was more than 50 per cent lower if tocopherol was not added (TABLE 2). The competitive inhibition of antimycin A

TABLE 2

EFFECT OF TOCOPHEROL AND ANTIMYCIN A ON REDUCTION BY DPNH OF CYTOCHROME c AND INDOPHENOL DYE USING DIGITONIN-SOLUBILIZED DPN-CYTOCHROME c REDUCTASE FROM RAT SKELETAL MUSCLE[27]

Treatment	*Cytochrome c reduction*	*2,6 Dichloro-indophenol reduction*
	ΔE_{550} *per 2 min.* $\times 10^3$	$-\Delta E_{635}$ *per 2 min.* $\times 10^3$
Isooctane-extracted enzyme		
Control	55	82
plus *d*-α-tocopherol	113	93
Unextracted enzyme		
Control	98	90
plus *d*-α-tocopherol	109	94
plus 0.001 μg. antimycin A	56	86
plus 0.002 μg. antimycin A	27	134
plus 0.004 μg. antimycin A	24	123
plus 0.01 μg. antimycin A	0	126

with tocopherol, and the lack of effect of isooctane extraction, tocopherol addition, and antimycin A when indophenol served as the electron acceptor suggest that the site of action of tocopherol is close to cytochrome c, probably between cytochromes *b* and c. That tocopherol is operating at catalytic levels was shown by experiments comparing the extent of DPNH oxidation with and without added tocopherol, using isooctane-extracted enzyme. After 90 min. the reaction mixture containing added tocopherol (17 mμmole) had oxidized 105 mμmole more of DPNH than had the corresponding control without added tocopherol. Thus, in this experiment 1 mμmole of added tocopherol promoted the oxidation of at least 6 mμmole of DPNH.[27]

Specificity of Tocopherol Stimulation

FIGURE 6a summarizes the effects of different concentrations of the tocopherols and derivatives, as well as other fat-soluble compounds, in restoring the activity of isooctane-extracted digitonin-solubilized DPN-

cytochrome c reductase. Except for differences in their affinities, the tocopherols and *d*-α-tocopheroxide were more or less equally effective, while tocopherylquinone and its hydroquinone (not shown) only partially restored enzyme activity. The succinate, acetate, disodium, phosphate, and polyethylene glycol 1000 succinate esters (not shown), as well as the vitamin E nucleus (2,2,5,7,8-pentamethyl, 6-hydroxychroman), gave little or no activity. Vitamin K_1, menadione, vitamin D_2, cardiolipin, cholesterol, oleic acid, and cystine had no effect (FIGURE 6b). Lipoic acid, cysteine, purified egg lecithin, and crude animal and vegetable lecithins were also inactive. The antioxidants Nordihydro-

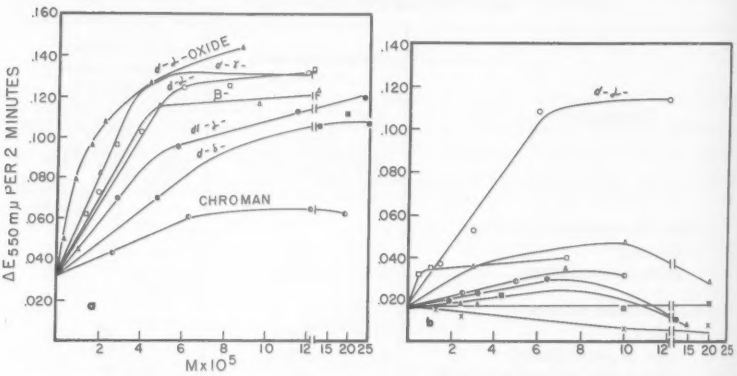

FIGURE 6. Effect of the tocopherols and other compounds in reactivating the digitonin-solubilized DPN-cytochrome c reductase from rat skeletal muscle.[27] (a) Represents the effects of the various tocopherols: ▲, *d*-α-tocopheroxide; □, *d*-γ-tocopherol; ○, *d*-α-tocopherol; △, *d*-β-tocopherol; ●, *dl*-α-tocopherol; ■, *d*-δ-tocopherol; ◐, 2,2,5,7,8-pentamethyl-6-hydroxychroman. (b) The effects of various other compounds: ○, *d*-α-tocopherol; □, vitamin K_1 (in ethanol); △, menadione (in 14 per cent acetone solution); ◐, cholesterol (in ethanol); ●, cardiolipin; ▲, vitamin D_2 (in ethanol); X, oleic acid (in alcohol); ■, cystine hydrochloride.

guairaetic acid, Santoflex B, Propylparasept, dibutyl p-cresol, diphenyl-p-phenylenediamine, and Santoquin in final concentrations ranging from 10^{-5} to 10^{-4} M showed no stimulating effect and, in the higher concentrations of the range, were in fact inhibitory. N-dodecyl aldehyde, an activator of luminescence by bacterial extracts[36] (final concentration 10^{-5} to 10^{-3} M), and the steroids pregnanediol, estrone, estradiol, cortisone E and cortisone F (10^{-6} to 5×10^{-6} M) were also without effect. Also ineffective were palmitic acid, tripalmitate, palmitic methyl ester, stearic acid, tristearate, stearic acid methyl ester, linolenic acid methyl ester, caprylic acid, capric acid, lauric acid, calcium phosphate gel, purified human γ-globulin, and egg albumin.[27] However, crystalline bovine serum albumin, in final concentrations of approximately 1 per cent, can

completely restore the activity of the isooctane-extracted enzyme. This has been shown to be due to a lipide material, bound to the albumin and extractable with isooctane, which is not free tocopherol.[28]

The activities of the different tocopherols in the cytochrome c reductase system show no correlation with their biological potencies as reported in the literature.[1-4] Neither Fe^{++}, Fe^{+++}, Cu^{++}, Zn^{++}, Co^{++}, Mn^{++}, Ca^{++}, BO_3^{---}, $MoO_4^{=}$, Mg^{++} (10^{-6} M to 5×10^{-4} M) nor a solution of the questionably small ash of tocopherol reactivated the extracted enzyme in place of added tocopherol.

Lipide "Cofactor" Extracted by Isooctane

In the absence of added tocopherol, restoration of enzyme activity has been obtained by adding to the extracted cytochrome c reductase the crude lipide residue remaining after vacuum distillation of the isooctane used in the extraction of the particulate enzyme from rat skeletal muscle or bovine heart. The crude lipide residue was significantly more effective than *d*-α-tocopherol in reactivating the enzyme; in most cases only 20 to 30 γ was required to restore 50 per cent of the activity of the extracted cytochrome c reductase, as compared to the 50 γ of *d*-α-tocopherol necessary for the same effect. Examination of such active, isooctane-extraction residues in quantities as high as 50 mg. in almost all cases failed to reveal the presence of any free tocopherol as measured by a paper chromatography procedure[37] with a demonstrated ability to detect 5 γ. Moreover, spectrophotometric examination of the residue before and after treatment with ascorbic acid, the latter to reduce any possible tocopheroxide present, showed no absorption in the region of 297 mμ characteristic of tocopherol. However, by using large quantities of enzyme (100 to 500 ml.) it was subsequently possible to show the presence of vitamin E in the lipide residue obtained by isooctane extraction. The recently described HCl-ascorbic acid reduction procedure[23] was used to convert a naturally occurring substance, presumably tocopherylquinone, to tocopherol. The amount of tocopherol, however, cannot account for enzymatic reactivation by the residue. For example, the quantity of crude lipide residue obtained by isooctane extraction of particulate rat skeletal muscle enzyme which restored 50 per cent of the activity of the extracted cytochrome c reductase contains only 0.03 γ of tocopherol even after HCl-ascorbic acid reduction. Since 45 to 50 γ of *d*-α-tocopherol are necessary for 50 per cent reactivation, the tocopherol content of the lipide residue is not responsible for enzymatic restoration. Instead, a lipide cofactor in the residue accounts for "reactivation."

The lipide cofactor in the crude lipide residue of the bovine heart muscle preparation obtained by extracting large quantities of the particulate enzyme with n-hexane was purified two hundredfold by alcohol fractionation and charcoal chromatography. The purified lipide is about

twenty times as active as tocopherol on a molar basis, and does not show the presence of tocopherol even after HCl-ascorbic acid treatment. Whereas the restoration activity of the lipide is completely destroyed by alkali hydrolysis and more than 50 per cent by acid, tocopherol is unaffected by either treatment. These findings further demonstrate that the reactivation effect of the crude lipide residue is not due to the presence of tocopherol, and that the normal activity of tocopherol, in turn, is not due to contamination by the lipide "cofactor." The purified lipide showed infrared bands characteristic of an ester linkage, and upon saponification it yielded glycerol as the alcohol component as characterized by conversion to the tribenzoate. The acid components were identified as stearic, palmitic, and oleic (in equimolar amounts) by partition chromatography and countercurrent distribution techniques.* These results as well as analytical and molecular weight data identified the lipide "cofactor" as a mixed triglyceride with stearate, oleate, and palmitate components.† That the cofactor is a mixed triglyceride and not a mixture of simple triglycerides was supported by the fact that mixtures of equimolar quantities and other combinations of tristearin, tripalmitin, and triolein failed to reactivate isooctane-extracted cytochrome c reductase. Chemical synthesis of the triglyceride produced material essentially identical to the lipide cofactor with respect to biological activity and other criteria.

The identification of the beef heart lipide cofactor as a mixed triglyceride stimulated a search for other lipides that might reactivate the isooctane-extracted enzyme. Of the more than one hundred different substances tested for restoration of activity, including steroids, mono-, di-, and triglycerides and other esters, phospholipide, fat-soluble vitamins and antioxidants, approximately ten showed significant restoration of activity. Some of these included milk, butter, oleomargarine, synthetic corn oil, *n*-butyl stearate, and certain synthetic glycerides (TABLE 3).

Interrelationship of Tocopherol and Active Lipides

That tocopherol is extracted from the enzyme by isooctane was shown by the presence of the vitamin in the nonsaponifiable fraction of the concentrated crude lipide residue obtained by isooctane extraction of large quantities of rat muscle and bovine heart muscle particulate cytochrome c reductase preparations. A significant portion of the vitamin is present as the presumed tocopherylquinone which can be reduced to tocopherol by the HCl-ascorbic acid procedure,[23] as already demonstrated by Bouman and Slater[38] for a Keilin-Hartree heart muscle preparation. The results of TABLE 4 show that only 3 to 17 per cent of the total tocopherol

*The countercurrent distribution studies were performed by John Coniglio, Vanderbilt University, Nashville, Tenn.

†K. O. Donaldson, A. Nason, I. R. Lehman, and A. Nikon. In preparation.

TABLE 3

ACTIVE SUBSTANCES FOR RESTORATION OF CYTOCHROME c REDUCTASE IN A RAT SKELETAL MUSCLE PARTICULATE PREPARATION

Substance	*γ for 50 per cent restoration*	*Percentage restoration*
d-α-Tocopherol	45 – 50	100
Lipide "cofactor"	7 – 10	100
n-Butyl stearate	19	95
Palmitolinolein	7	82
Cream	29	43
Synthetic corn oil	11	82
Pet milk	19	66
Oleomargerine	20	70
Butter	17	31
Ethyl myristate	58	59
1,3-Diolein	29	72

TABLE 4

TOCOPHEROL CONTENT OF PARTICULATE ENZYME PREPARATIONS FROM RAT SKELETAL MUSCLE AND BOVINE HEART MUSCLE[30] (μM TOCOPHEROL/GM. PROTEIN)

Preparation	*Isooctane-extracted*		*Remaining*		*Total*
	Before reduction	*After HCl-ascorbic acid reduction*	*Before reduction*	*After HCl-ascorbic acid reduction*	
Rat skeletal muscle enzyme:					
1	0.18	0.42	0.82	4.08	4.5
2	0.08	0.16	0.75	4.94	5.1
3	0.18	0.90	1.55	4.30	5.2
Bovine heart muscle enzyme:					
1	1.33	1.60	4.44	8.40	10.0
2	1.88	1.65	5.68	9.65	11.3
3	1.55	1.57	3.70	8.43	11.0

of the rat skeletal muscle enzyme is removed by isooctane extraction with a concomitant loss of approximately 90 per cent of the cytochrome c reductase activity. The bovine heart muscle system contains twice as much vitamin E as the rat skeletal enzyme, with a larger ratio of tocopherol to presumed quinone. Most of the vitamin E removed from the bovine system is in the α-tocopherol form. Tocopherol was determined colorimetrically by a modification of the iron chloride-bipyridyl method of

Emmerie and Engel[39] and confirmed to be α-tocopherol by the paper chromatographic procedure of Green *et al.*[40]

The fact that structurally dissimilar molecules such as the tocopherols and certain glycerides and fatty acid esters reactivate isooctane-extracted particulate cytochrome c reductase, which still has 90 per cent of its original vitamin E, suggested the following relationship. Although the small quantity of tocopherol in the crude lipide residue cannot account for enzymatic restoration, it is possible that the tocopherol was originally present at the "active enzyme sites," before isooctane extraction. Addition of the lipide cofactor or of certain other effective esters might replenish the "active sites" of the extracted enzyme by releasing to them some of the remaining tocopherol, perhaps from a "bound" form. This would be the same as adding tocopherol to the system. The activity of these effective esters could thus be attributed directly to tocopherol. As a partial test of this hypothesis an examination was made of the effects of various lipides in potentiating the removal of tocopherol from the enzyme by isooctane extraction. TABLE 5 shows that the addition of the lipide cofactor or of the indicated active substances to the bovine heart muscle enzyme followed by isooctane extraction results in

TABLE 5

EFFECT OF VARIOUS LIPIDES ON THE SUBSEQUENT RELEASE OF TOCOPHEROL BY ISOOCTANE EXTRACTION OF BOVINE HEART ENZYME[30]

Substances	*Effect in restoration of isooctane-extracted enzyme*	*μM of tocopherol extracted*	
		Before reduction	*After HCl-ascorbic acid reduction*
EtOH-albumin (control)	Inactive	0.07	0.33
Glycerol dipalmitate	Inactive	0.09	0.36
Tristearin	Inactive	0.09	0.35
Purified lipide "cofactor" (from bovine heart muscle)	Active	0.16	0.86
Oleomargarine	Active	0.27	1.09
n-Butylstearate	Active	0.14	0.65

a two- to threefold release of tocopherol and presumed tocopherylquinone, as compared with the control. Inactive compounds such as glycerol dipalmitate and tristearin were ineffective in potentiating the removal of tocopherol by subsequent isooctane extraction. Other inactive substances such as glycerol monostearate, lauric acid, and tripalmitin (not shown) were also ineffective, whereas butter and ethyl stearate (not shown), which have restoration power, resulted in a three- to fivefold greater removal of vitamin E from the enzyme.

TABLE 6 shows that the progressive removal of vitamin E by increasing number of isooctane extractions is accompanied by a corresponding decrease in cytochrome c reductase activity. If the lipide cofactor is added after six extractions and if then three more extractions are performed, a greater amount of tocopherol is removed than from the extracted control. Morrison *et al.*,[41] using isooctane-extracted succinate-cytochrome c reductase from pig heart, have recently confirmed the reactivating effect of *d*-α-tocopherol and the lipide residue derived from isooctane extraction. Reactivation was also effected by a hemin-lipide complex isolated from heart muscle. It is not unlikely that the hemin-lipide might be acting like the other active compounds in potentiating the mobility of endogenous tocopherol as suggested above.

Tocopherol Specific Cytochrome c Reductase

Vitamin K_1 was one of the substances that did not restore the isooctane-extracted, digitonin-solubilized cytochrome c reductase (FIGURE 6b). With the isooctane-extracted particulate enzyme, however, it was fully active. Upon digitonin-solubilization of the particulate enzyme, vitamin K_1 no longer had a restoration effect. Vitamin K_1 could be acting indirectly by potentiating the movement of the endogenous tocopherol of the particulate preparation to the "active sites" of the enzyme. Perhaps digitonin solubilization resulted in a loss of sufficient tocopherol from the enzyme, the vitamin K_1 having no potentiation effect on the remaining endogenous tocopherol. The lipide cofactor and other effective substances, however, still reactivated. That an even narrower specificity for restoration by tocopherol might be attained by further dissociation of endogenous tocopherol from the enzyme seemed likely. This has been accomplished by aging particulate or digitonin-solubilized enzyme preparations of rat skeletal muscle or bovine heart muscle for about 2 weeks at 0° or for 2 to 5 days at -15° C. with daily thawing. Particulate enzyme preparations homogenized with 2 per cent aqueous digitonin suspensions instead of the usual 4 per cent[25,26] best withstood the above storage conditions. The lowered succinate-cytochrome c reductase activity of a stored, isooctane-extracted digitonin-solubilized bovine heart preparation was fully and specifically restored by *d*-α-tocopherol (FIGURE 7). The lipide cofactor *n*-butyl stearate, oleomargarine, butter, vitamin K_1, milk and cream, hitherto demonstrated to reactivate particulate and freshly digitonized preparations, were ineffective. The β and γ-tocopherol (not shown) were as effective as tocopherol; other tocopherol compounds, for example, the hydroquinone and tocopheroxide, were partially (50 per cent) active and the quinone was totally inert. It is possible that the partial restoration effect of the hydroquinone and tocopheroxide may be due to a potentiation effect, and that with even further dissociation of endogenous tocopherol from the enzyme, these compounds will no longer

TABLE 6

RELATIONSHIP OF CYTOCHROME C REDUCTASE ACTIVITY IN PARTICULATE PREPARATIONS TO VITAMIN E CONTENT OF THE ENZYME

No. of extractions	*Cytochrome c reductase activity* ($\Delta E_{550m\mu}/2'$)			*Percentage free tocopherol removed*	*Percentage total tocopherol removed*
	Control	*plus α-tocopherol*	*plus lipide "cofactor"*		
Beef heart					
0	142	180	160	–	–
3	72	153	130	6.4	15.2
6	39	121	100	7.6	23.2
9	5	82	41	9.0	31.9
9 + "cofactor"	0	38	8	11.7	47.0
12	0	68	39	12.8	54.1
Rat muscle					
3	18	122	111	2.0	
6	12	118	110	3.0	
6 + "cofactor"	11	79	69	6.7	

reactivate. Similar data have been obtained with the bovine DPN-cytochrome c reductase and the DPN-and succinate systems of rat muscle. That isooctane was not acting as an inhibitor whose effect could be reversed by tocopherol was shown by experiments in which isooctane was not employed. Instead, inactivated enzymes were obtained by aging the particulate or digitonized bovine enzymes for 1 to 2 wk. at -15° with daily thawing. These enzymes were then fully and specifically restored by the tocopherols.

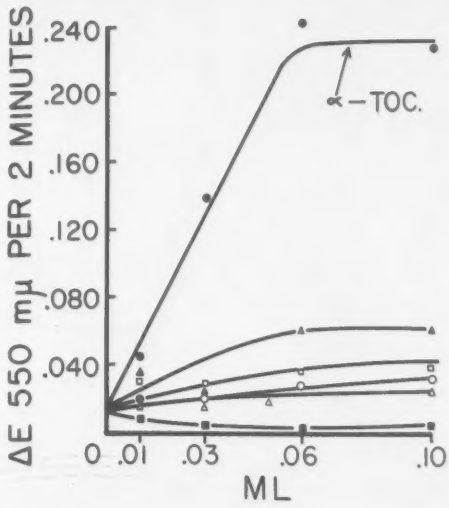

FIGURE 7. Effect of *d*-α-tocopherol and other substances in reactivating aged digitonin-solubilized bovine heart muscle succinate-cytochrome c reductase after isooctane extraction. The various substances (5 mg./ml.) were added in 15 per cent ethanol-0.2 per cent albumin: ●—●, *d*-α-tocopherol; ▲—▲, butter; □—□, *n*-butyl stearate; ○—○, oleomargarine; △—△, purified lipide "cofactor"; ■—■, vitamin K_1.

The lipide cofactor and *n*-butyl stearate, which are ineffective in restoring cytochrome c reductase activity of the above tocopherol-specific enzymes, do not potentiate the extractability by isooctane of endogenous tocopherol from these preparations. They do, however, release the presumed tocopherylquinone; but, as already shown, the quinone is completely ineffective in reactivating these systems. Preliminary experiments demonstrate that progressive aging of the preparations results in a correspondingly greater dissociation of endogenous tocopherol from the enzyme. This in turn is accompanied by a more specific tocopherol restoration effect.

Abolition of Lag by Tocopherol

An initial lag period before maximal reactivation of isooctane-extracted pig heart succinate-cytochrome c reductase by *d*-α-tocopherol[41] also occurs with the bovine and rat muscle DPN- and succinate-cytochrome c reductases. The lag, however, can be abolished progressively with increasing periods of preincubation of the enzyme with *d*-α-tocopherol (FIGURE 8). A preincubation period of approximately 10 min. is necessary for maximal activation as measured at the half-minute reading (Inset Curve, FIGURE 8). β- and γ-tocopherols are equally effective.

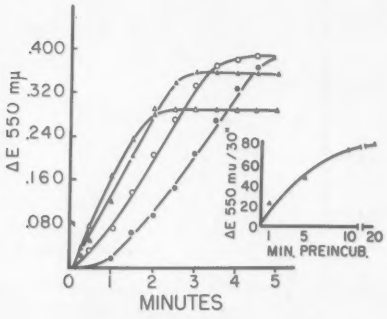

FIGURE 8. Time course of restoration of aged digitonin-treated isooctane-extracted bovine heart muscle succinate-cytochrome c reductase after different times of preincubation of enzyme with *d*-α-tocopherol.[31] Inset curve: effect of time of preincubation of tocopherol and digitonin-treated bovine heart muscle succinate-cytochrome c reductase on the enzymatic reaction at the 30 sec. reading.[31] The optical density units in the inset curve are expressed as Log $\frac{I_o}{I} \times 10^3$.

Conclusion

The above results strongly implicate tocopherol as one of the active components of the terminal respirating chain in mammalian skeletal and heart muscle tissue. It appears to be functioning as a cofactor in the cytochrome c reductase portion of both the DPN oxidase and succinate oxidase systems just before the level equivalent to cytochrome c as shown in the following scheme of electron transfer:

$$\text{DPNH} \rightarrow \text{flavin (?)} \rightarrow \text{cytochrome } b \xrightarrow{\text{tocopherol}} \text{cytochrome } c_1 \text{ and } c \rightarrow a \rightarrow a_3 \rightarrow O_2$$

or
(succinate)

DPN-cytochrome c reductase and succinate-cytochrome c reductase have been shown to be separate systems in the present study, although

they are indicated together in the above sequence for purposes of convenience. The data are not conclusive that cytochromes *b*, c_1, and *c* are components on the main pathway of the above system in rat skeletal muscle, nor is there as yet definite information that a flavin is involved. Evidence in favor of a role of *d*-α-tocopherol as a specific cofactor in the DPN- and succinate-cytochrome c reductases includes the following: (1) tocopherol is present in rat skeletal muscle and bovine heart muscle preparations[30,38] in amounts commensurate with those of other established cofactors (for example, cytochrome *c*); (2) isooctane extraction, or aging accompanied by freezing and thawing, inactivates cytochrome c reductase with a concomitant loss or dissociation of vitamin E from the enzyme; and (3) enzymatic activity is specifically restored by the tocopherols.

The earlier observations with relatively fresh enzyme preparations that such structurally unlike molecules as tocopherol and certain triglycerides and long chain fatty acid esters can also restore cytochrome c reductase activity have been reconciled with the ability of the latter substances to release "bound" endogenous tocopherol to the "active sites" of the enzyme. Direct evidence for a specific tocopherol effect was obtained with aged enzyme preparations whose activity was restored only by the tocopherols, the above neutral fats having no effect on reactivation or release of "bound" endogenous tocopherol. Although *d*-α-tocopherol has been shown to function in catalytic amounts, it has not been possible thus far to demonstrate that it undergoes alternate oxidation and reduction. Tocopherol will not serve as an electron donor in place of DPNH or succinate in the enzymatic reduction of cytochrome c. Neither will tocopheroxide act as an electron acceptor for DPNH oxidation in this system. In view of the limitation of detecting oxidation-reduction changes of small quantities of tocopherol bound to the enzyme, a role for tocopherol as an electron carrier has not been ruled out. The ineffectiveness of the quinone and the partial activity of tocopheroxide would suggest that these two oxidation forms are not involved. Perhaps tocopherol functions in electron transport by undergoing a reversible oxidation change to its free radical state.[22] The elimination of the initial lag in reaction rate by preincubation with tocopherol indicates that this is the time necessary for either (1) activation of tocopherol to some coenzyme form, or (2) association of tocopherol with the enzyme.

Although the role of the vitamin as an electron carrier is favored, the alternate possibility that it is functioning as a binding material has not been eliminated. Tocopherol might conceivably act as a cementing material or part of a lipide sheath that maintains cytochrome *b* and c in a juxtaposition or spatial configuration for optimal reaction with each other. The importance of spatial integration in the functioning of the succino-oxidase complex has been stressed by Keilin and Hartree,[42] who attributed the marked increase in activity of their preparations by protein and calcium phosphate gel to a nonspecific effect on the colloidal

structure of the enzyme, presumably by reorienting the catalytic components, especially endogenous c, with the colloidal particles. Lysine and glyoxaline-4,5-dicarboxylic acid,[43] and histidine[44] have also been shown to activate preparations of Keilin-Hartree horse heart succinate oxidase in unfavorable environments. Altman and Crook,[45] however, have ascribed the action of the above materials as well as other chelating agents including versene to their ability to adsorb or chelate heavy metals that are present as contaminants in the reaction mixture. The results reported here with tocopherol are different from those reported in the above literature. The strikingly high lipide content of partially purified oxidases of DPNH and succinate ranging from 30 to 60 per cent on a dry weight basis are made up of various constituents. Lecithinase has been reported[46-50] to inactivate liver and heart terminal electron transport systems near the site of reduction, as well as oxidation of cytochrome c, although there was no reported attempt to reactivate by readding lecithin. In view of the inactivation of the system by various phospholipases, Edwards and Ball[46] feel that the function of phospholipides in succinate oxidase is that of a cementing substance. They used succinate oxidase preparations containing 25 to 30 per cent phospholipide, one half of which was lecithin, the other half being composed of phosphatidyl ethanolamine and phosphatidyl serine with negligible amounts of sphingomyelin, cardiolipin, and acetal phosphatides.

It is also possible that α-tocopherol may be functioning as a component of the phosphorylating respiratory chain, perhaps as a link between respiration and oxidative phosphorylation. TPNH oxidation in liver mitochondria does not yield energy-rich phosphate bonds,[51] and the TPN-cytochrome c reductase of rat skeletal muscle, although lowered by isooctane extraction, is not restored by the addition of tocopherol.[27] During the last two years there have been reports of a decrease of oxidative phosphorylation in cell-free preparations from vitamin E-deficient tissues. Martius[52] observed that particles isolated from the diaphragm of vitamin E-deficient animals had a lowered P:O ratio. He envisaged α-tocopherol as a cofactor in oxidative phosphorylation at the level between cytochromes *b* and c. McCay and Caputto[53] also found a diminished efficiency of oxidative phosphorylation in liver mitochondria isolated from vitamin E-deficient rats. Their failure to localize the site of uncoupling raised doubts as to whether this was a specific tocopherol effect or was due to a nonspecific damage of the liver mitochondria. This, of course, raises the question as to whether or not deficiencies of other nutritional factors, organic or inorganic, as well as various pathological conditions would result in a lowered P:O ratio. Weinstock and his associates[54] observed an increase in oxidation of tricarboxylic acid substrates by washed liver homogenates with added adenosine triphosphate. They speculated that vitamin E deficiency may affect yet another mechanism controlling oxida-

tion that is independent of the levels of phosphate-bond acceptor systems.

Recent investigations by others have uncovered additional new phases of vitamin E metabolism. Dinning and Day and their co-workers[55-58] have shown that vitamin E-deficient rabbits excrete extra quantities of allantoin[55] and that the livers exhibited greatly increased xanthine oxidase activities.[56] Vitamin E deprivation in rabbits led to a greatly increased incorporation of formate into nucleic acid purines of both ribonucleic acid and deoxyribonucleic acid of skeletal muscle and bone marrow.[57] Although there was less glycine incorporated into nucleic acids and proteins,[57] there was an increased rate of creatine synthesis and an increased turnover of skeletal muscle creatine.[58] It was suggested that vitamin E may control the exchange of the carbon of the 2 position of the purine ring, possibly through an effect on the synthesis or activity of coenzymes derived from folic acid. Simon *et al*[59] have discovered 2 new metabolites of α-tocopherol, largely in conjugated form, in the urines of humans ingesting large quantities of vitamin E. These were identified as 2-(3-hydroxy-3-methyl-5-carboxypentyl)-3,5,6-trimethylbenzoquinone and its γ-lactone. Rodnan, Chernick, and Schwarz[60] found both α- and γ-tocopherol to be quite potent, upon intraportal injection, in their ability to reverse the respiratory decline of oxygen consumption of histologically unaffected liver slices of rats during the latent phase of nutritionally induced necrotic liver degeneration. β- and δ-tocopherol were much less effective. Hager[61] has recently reported that α-tocopherol phosphate and α-tocopherol succinate were able to replace a naturally occurring factor or factors in the enzymatic ferricyanide-linked oxidative decarboxylation of pyruvate to acetate and CO_2 catalyzed by a purified soluble pyruvate oxidase from an acetate-requiring mutant of *Escherichia coli*.

There is little doubt that eventual elucidation of the mechanism(s) of action of vitamin E at the enzymatic level as well as its secondary activity as an antioxidant will ultimately serve to explain its various effects in biological processes.

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SECTION OF MATHEMATICS AND ENGINEERING

TRANSIENT TEMPERATURES IN NUCLEAR REACTORS COOLED BY LIQUIDS*

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Abstract

Expressions are derived for transient temperatures of wall and fluid in a source-containing channel cooled by a liquid. The structure of the general solution is briefly noted; numerical results are presented for the special case of zero source strength.

The temperature solutions apply to warm-up, start-up and, under some conditions, power-change of core-moderated reactors and provide a basis for calculation of thermal stress and temperature-dependent nuclear reactivity.

Nomenclature

The following nomenclature is used in the paper:

A	coefficient in expression for source strength, dimensionless
a	channel flow area, ft.^{2}
B	coefficient in expression for source strength, dimensionless
C	coefficient in expression for source strength, Btu/sec. ft.^{3}
c	specific heat, Btu/lb. °F.
D	coefficient in expression for source strength, Btu/sec. ft.^{3}
e	base of natural logarithms, dimensionless
$F_1(\tau)$, $F_2(\tau)$	functions of τ defined by EQUATIONS 44 and 45, dimensionless
H	unit step function, H(argument) = 0 for argument ≤ 0, H(argument) = 1 for argument > 0, dimensionless
h	coefficient of convective heat transfer, Btu/sec. ft.^{2} °F.
I_0	modified Bessel function of first kind of zero order, dimensionless
k	coefficient defined by EQUATIONS 34 and 35, °F.
L	total length of channel, ft.
$\mathcal{L}$	Laplace Transform of a function of time
M	weight, lb.
n	coefficient defined by EQUATIONS 37 and 38, °F.
p	argument of the Laplace Transform, sec.$^{-1}$

*This paper was presented at a meeting of the Section on October 18, 1957.

q'''	strength of volumetric wall source, Btu/sec. ft.^{3}
r_1, r_2	coefficients of τ in exponents of expression for source strength, sec.$^{-1}$
$r_{3,4,5,6}$	factors of the fourth degree polynomial given in EQUATION 33, sec.$^{-1}$
S	total heat transfer surface in channel, ft.^{2}
T	fluid temperature, °F.
$\overline{T}_{1,2,3}$	functions of p defined in EQUATIONS 25, 26, 27, °F. sec.
t	wall temperature, °F.
$\overline{\tau}_{1,2,3,4}$	functions of p defined in EQUATIONS 53, 54, 55, 56, °F. sec.
v	fluid velocity, ft./sec.
W	fluid weight flow, lb./sec.
x	distance from channel entrance, ft.

Greek

a	$hS/[L(\rho c)_f]$ sec.$^{-1}$
β	$hS/(Mc)_w$ sec.$^{-1}$
ϵ	$t_o\left(1+\frac{a}{p+\beta}\right)/v$ °F. sec./ft.
η	$p\left(1+\frac{a}{p+\beta}\right)/v$ ft.$^{-1}$
μ	$\frac{a}{v}\ \frac{1}{(\rho c)_w}\ \left[\frac{C}{(p+\beta)(p-r_1)}+\frac{D}{(p+\beta)(p-r_2)}\right]$ °F. sec./ft.
ξ	argument of integration, sec.
ρ	density, lb./ft.^{3}
σ	argument of integration, sec.
τ	time, sec.
ϕ	$(\tau-\frac{x}{v})$, sec.
ψ	argument of integration, sec.

Subscripts

f	fluid
j	sequential integer identifying a member of a set
o	initial or entrance
w	wall

Superscript

bar (−)	Laplace Transform of a function of τ

Introduction and Statement of Problem

A substantial literature exists on the determination of fluid and channel wall temperatures in ducted heat exchanging flows in the transient state. Carslaw and Jaeger[1] summarize and extend early studies on systems in which heat sources are absent, and show that when the equations of

transient convective heat transfer are linear they are often readily integrated by use of the Laplace Transformation. Clark, Arpaci, and Treadwell[2a] list more recent papers, a number of which treat problems involving heat sources, and themselves solve for the fluid and wall temperatures in a channel containing a spatially uniform wall source that undergoes a step change in strength after prior steady operation.

This paper is concerned with transient fluid and wall temperatures in a channel containing a wall source of strength that varies in both time and location. In the system studied, the liquid coolant and the duct wall are initially at the same temperature everywhere; then, at some instant of time, the wall source starts to generate heat according to the relation

$$q''' = \sin\left(A - B\frac{x}{L}\right)\left[Ce^{r_1\tau} + De^{r_2\tau}\right] \qquad (a)$$

and simultaneously the coolant undergoes step changes in its flow rate and inlet temperature. This problem might arise under several circumstances in a channel of a core-moderated end-reflected reactor, depending on the values assigned to the constants in EQUATION *a*. If

$$A = B = 0, \text{ or } C = D = 0 \qquad (b)$$

the description of a source-free system is obtained and is applicable to the reactor warm-up period prior to the start of power generation; while if D and r_2 are assigned properly averaged values,

$$D < 0, \; r_2 < 0 \qquad (c)$$

reactor start-up is described.[3] The solution obtained under the condition shown in EQUATION c would also apply with good approximation to a reactor that has been operating at very low (nominally zero) power, with sufficient coolant flow so that the fluid and channel wall temperatures are at essentially the same uniform temperature, if the nuclear reactivity and the fluid flow and inlet temperature are suddenly increased.

Other applications are obtainable from the general solution based on EQUATION *a* if appropriate values are assigned in the solution to the constants of this equation. For example, if

$$r_1 = r_2 = 0 \qquad (d)$$

the general solution is specialized to that for a step change in source strength from a zero initial value to a function truncated sinusoidally in space.*

Following Carslaw and Jaeger,[1] the Laplace Transformation is employed for integrating the differential equations, and analytical solutions are obtained for the coolant and wall temperatures in the general case

*By Descartes' Rule of Signs.

described by EQUATION *a*. The significance of the form of the general solution is indicated briefly. Numerical results are given for the special case of zero source strength; these numerical values extend those published earlier by Schumann[4] and would be useful for reactor warm-up calculations.

The analysis reported here was performed in the Clifton, N. J., branch of the Curtiss-Wright Research Division's Nuclear Department. Use of the Curtiss-Wright IBM-704 to generate numerical results for the case of zero source strength, and the assistance of J. DeVries and G. Beveridge in programming the calculations are gratefully acknowledged.

ANALYSIS

Assumptions

The following simplifying approximations are employed:

(1) The coolant is a liquid moving in slug flow; its velocity and temperature are uniform across every section, and its physical properties are constant.

(2) The channel cross-sectional area is constant along the channel length; the channel wall temperature at any section is uniform across the wall thickness; and the outer surface of the wall exchanges no heat with the exterior.

(3) The convective heat-transfer coefficient has the same value everywhere in the channel, and axial conduction is negligible both in the wall and fluid.

(4) Initially the coolant and channel wall are at the same temperature everywhere and no heat is generated in the wall; at the start of the transient state the coolant experiences step changes in flow rate and inlet temperature, while the wall source starts to generate heat according to EQUATION *a* of the *Introduction*.

Heat Balance Equations, Initial and Boundary Conditions

For the stipulated assumptions, the heat balance for an elementary volume of the duct wall is

$$\frac{\partial t}{\partial \tau} = \frac{q'''}{(\rho c)_w} - \frac{hS}{(Mc)_w}(t - T) \equiv \frac{q'''}{(\rho c)_w} - \beta (t - T) \qquad (1)$$

which states that in the absence of axial conduction and heat loss to the exterior, the rate of change of wall temperature at any instant is governed solely by the difference between the rate at which heat is generated within the wall and the rate at which heat is transferred to the fluid. The initial condition for the wall temperature at the arbitrary point x is

$$t(x, 0) = t_o \qquad (2)$$

and the source term q''' is given, in the present study, by

$$q''' = \sin\ (A - B\tfrac{x}{L})\ [\ Ce^{r_1\tau} + De^{r_2\tau}\ ] \tag{3}$$

The heat balance for an elementary volume of fluid is given in Carslaw and Jaeger[1] (Equation 1, p. 326); in the present notation

$$\frac{\partial T}{\partial \tau} = \frac{hS}{L\mathcal{A}(\rho c)_f}\ (t-T) - \frac{W}{\mathcal{A}\rho_f}\ \frac{\partial T}{\partial x} \equiv \alpha\ (t-T) - v\ \frac{\partial T}{\partial x} \tag{4}$$

which implies that in the absence of axial conduction, the rate of change of temperature in the elementary volume $\mathcal{A}dx$ at x is governed by the difference between the rate at which heat is transferred into the volume by the wall and the net rate at which enthalpy is convected into and out of the volume as the fluid flows through. The initial condition for the arbitrary point x is

$$T(x,0) = t_o \quad \text{(assumption 4)} \tag{5}$$

while at the entrance where the step change occurs at $\tau = 0$,

$$T(0,0) = T_o \tag{6}$$

$$T(0,\tau) = T_o \tag{7}$$

Transformation of Equations

Familiarly, the Laplace Transformation of a function of τ is accomplished by multiplying the function by $e^{-p\tau}d\tau$ and integrating the product between the limits $\tau = 0$ and $\tau = \infty$. Applying this transformation to EQUATIONS 1, 3, 4, these equations take the forms

$$\overline{\left(\frac{\partial t}{\partial \tau}\right)} = \frac{\overline{q'''}}{(\rho c)_w} - \beta\ (\bar{t} - \bar{T}) \tag{8}$$

$$\left.\begin{aligned} \overline{q'''} &= \sin\ (A - B\tfrac{x}{L})\ \int_o^\infty [Ce^{-(p-r_1)\tau} + De^{-(p-r_2)\tau}]\,d\tau \\ &= \sin\ (A - B\tfrac{x}{L})\left[\frac{C}{p-r_1} + \frac{D}{p-r_2}\right] \end{aligned}\right\} \tag{9}$$

in which p has been taken positive and sufficiently large that the quantities $(p-r_1)$ and $(p-r_2)$ are both positive numbers; also

$$\overline{\left(\frac{\partial T}{\partial \tau}\right)} = \alpha\ (\bar{t} - \bar{T}) - v\ \overline{\left(\frac{\partial T}{\partial x}\right)} \tag{10}$$

Now

$$\left(\overline{\frac{\partial t}{\partial \tau}}\right) = p\bar{t} - t(x,0) \qquad (11)$$

$$\left(\overline{\frac{\partial T}{\partial \tau}}\right) = p\bar{T} - T(x,0) \qquad (12)$$

} Carslaw and Jaeger[1] (Theorem II, p. 241).

and

$$\left(\overline{\frac{\partial T}{\partial x}}\right) = \frac{\partial \bar{T}}{\partial x} \quad \text{Carslaw and Jaeger[1] (Theorem III, p. 241).} \qquad (13)$$

Using EQUATIONS 11 through 13, together with EQUATIONS 2 and 5, EQUATIONS 8 and 10 become

$$p\bar{t} - t_o = \frac{\overline{q'''}}{(\rho c)_w} - \beta(\bar{t} - \bar{T}) \qquad (14)$$

$$p\bar{T} - t_o = a\bar{t} - a\bar{T} - v\frac{\partial \bar{T}}{\partial x} \qquad (15)$$

Solving for $\bar{t}$ in 14 and substituting 9 for q''',

$$\bar{t} = \frac{t_o}{p+\beta} + \left(\frac{\beta}{p+\beta}\right)\bar{T} + \frac{\sin(A - B\frac{x}{L})}{(\rho c)_w}\left[\frac{1}{p+\beta}\left(\frac{C}{p-r_1} + \frac{D}{p-r_2}\right)\right] \qquad (16)$$

Substituting 16 into 15, and re-arranging,

$$\frac{\partial \bar{T}}{\partial x} + \frac{p}{v}\left(1 + \frac{a}{p+\beta}\right)\bar{T} = \frac{t_o}{v}\left(1 + \frac{a}{p+\beta}\right) + \frac{a}{v}\frac{\sin(A - B\frac{x}{L})}{(\rho c)_w}\left[\frac{1}{p+\beta}\left(\frac{C}{p-r_1} + \frac{D}{p-r_2}\right)\right] \qquad (17)$$

Using the symbols η, ϵ and μ defined in the *Nomenclature*, 17 becomes

$$\frac{\partial \bar{T}}{\partial x} + \eta\bar{T} = \epsilon + \mu \sin(A - B\frac{x}{L}) \qquad (18)$$

which is an ordinary linear equation of first order having $e^{\eta x}$ as integrating factor and integrates straightforwardly to

$$\bar{T} = (\text{constant})\, e^{-\eta x} + \frac{\epsilon}{\eta} + \frac{\mu\eta \sin(A - B\frac{x}{L}) + \mu(\frac{B}{L})\cos(A - B\frac{x}{L})}{\eta^2 + (\frac{B}{L})^2} \qquad (19)$$

The constant in 19 is determined by use of the fluid entrance and initial conditions shown in 7 and 6, together with the general relation 12. EQUATION 7 yields

$$\frac{\partial T(0,\tau)}{\partial \tau} = 0 \qquad (20)$$

and

$$\left[\overline{\frac{\partial T(0,\tau)}{\partial \tau}}\right] = 0 \tag{20a}$$

Substituting 20a into 12 and using 6 for $T(0,0)$

$$0 = p\overline{T}(0,p) - T_o \tag{21}$$

Thus

$$\overline{T}(0,p) = \frac{T_o}{p} \tag{22}$$

Substituting 22 into 19, setting $x = 0$ and using the fact that $\frac{\epsilon}{\eta} = \frac{t_o}{p}$,

$$\frac{T_o}{p} = \text{constant} + \frac{t_o}{p} + \frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2}$$

which yields

$$\text{constant} = \frac{T_o - t_o}{p} - \frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2} \tag{23}$$

and substituting into 19 and rearranging

$$\overline{T} = \left[\frac{t_o}{p} + \left(\frac{T_o - t_o}{p}\right) e^{-\eta x}\right] + \left[\frac{\mu\eta \sin\left(A - B\frac{x}{L}\right) + \mu\left(\frac{B}{L}\right)\cos\left(A - B\frac{x}{L}\right)}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] - \left[\frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] e^{-\eta x} \tag{24}$$

Let

$$\overline{T_1} \equiv \left[\frac{t_o}{p} + \left(\frac{T_o - t_o}{p}\right) e^{-\eta x}\right] \tag{25}$$

$$\overline{T_2} \equiv \left[\frac{\mu\eta \sin\left(A - B\frac{x}{L}\right) + \mu\left(\frac{B}{L}\right)\cos\left(A - B\frac{x}{L}\right)}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] \tag{26}$$

$$\overline{T_3} \equiv -\left[\frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] e^{-\eta x} \tag{27}$$

Then

$$\overline{T} = \overline{T_1} + \overline{T_2} + \overline{T_3} \tag{28}$$

Solution for Fluid Temperature

The fluid temperature as function of x and τ is obtained by inverting $\overline{T}$. This may be done by inverting $\overline{T}_1$, $\overline{T}_2$, $\overline{T}_3$ separately and adding the results.

(1) *Inverse of* $\overline{T}_1$. $\overline{T}_1$ is the transform of the fluid temperature in a source-free channel. This case is treated operationally by Carslaw and Jaeger,[1] (p. 326), hence the inverse of $\overline{T}_1$ can be taken directly from that reference. Before citing that inverse, the second term in $\overline{T}_1$ is first put explicitly in terms of p for later use.

$$\frac{1}{p}e^{-\eta x} \equiv \frac{1}{p}e^{-\frac{px}{v}}\left[1+\frac{\alpha}{p+\beta}\right] = \frac{1}{p}e^{-\frac{px}{v}}\;e^{-\frac{\alpha x}{v}}\left(\frac{p}{p+\beta}\right)$$

$$= \frac{1}{p}e^{-\frac{px}{v}}\,e^{-\frac{\alpha x}{v}}\left(1-\frac{\beta}{p+\beta}\right) = \frac{1}{p}e^{-\frac{\alpha x}{v}}\,e^{-\frac{px}{v}}\;e^{\frac{\alpha\beta x}{v(p+\beta)}} \tag{29}$$

Multiplying the right member of 29 by $(p+\beta)/(p+\beta)$ and rearranging $(p+\beta)/p$ as $(1+\beta/p)$,

$$\frac{1}{p}e^{-\eta x} = e^{-\frac{\alpha x}{v}}\left[e^{-\frac{px}{v}}\frac{e^{\frac{\alpha\beta x}{v(p+\beta)}}}{p+\beta} + \beta\,\frac{e^{-\frac{px}{v}}}{p}\;\frac{e^{\frac{\alpha\beta x}{v(p+\beta)}}}{p+\beta}\right] \tag{30}$$

Referring now to Carslaw and Jaeger for inverses of terms in 30, and applying to 25,

$$T_1 = t_o + H(\phi)(T_o - t_o)\,e^{-\frac{\alpha x}{v}}\left[e^{-\beta\phi}I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) + \beta\int_0^{\phi} e^{-\beta\psi}\;I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\psi}\right)d\psi\right] \tag{31}$$

in which $\phi \equiv (\tau - \frac{x}{v})$ and the presence of the unit step function $H(\phi)$ signifies that 31 is inapplicable until $\phi > 0$, that is, until enough time has elapsed so that the fluid front, moving at velocity v, has had an opportunity to reach the point x.

(2) *Inverse of* $\overline{T}_2$. To invert $\overline{T}_2$ it is necessary to invert the terms $\frac{\mu\eta}{\eta^2+(\frac{B}{L})^2}$ and $\frac{\mu}{\eta^2+(\frac{B}{L})^2}$; functions of x alone are treated the same as constants. Referring to the *Nomenclature*,

$$\frac{\mu\eta}{\eta^2+(\frac{B}{L})^2} = \frac{\alpha}{(\rho c)_w}\;\frac{p(p+\alpha+\beta)}{p^2(p+\alpha+\beta)^2+(\frac{Bv}{L})^2(p+\beta)^2}\left[\frac{C}{p-r_1} + \frac{D}{p-r_2}\right] \tag{32}$$

Now,

$$p^2(p+\alpha+\beta)^2 + (\tfrac{Bv}{L})^2(p+\beta)^2 \equiv p^4 + p^3\,[2\alpha + 2\beta]$$

$$+ p^2\,[(\alpha+\beta)^2 + (\tfrac{Bv}{L})^2] + p\,[2\beta(\tfrac{Bv}{L})^2] + \beta^2(\tfrac{Bv}{L})^2 \qquad (33)$$

$$\equiv (p-r_3)(p-r_4)(p-r_5)(p-r_6)$$

in which r_3, r_4, r_5 and r_6 may be negative* or complex, but for definiteness in the following development it will be assumed that they are all different from one another. (If two or more of the factors are the same, the development would have to be modified somewhat from the following.) If 33 is substituted into 32 the resulting function is expressible as the sum of partial fractions,

$$\begin{aligned}
\frac{\mu\eta}{\eta^2+(\frac{B}{L})^2} = {} & \frac{1}{p-r_1}\left[\frac{\alpha C}{(\rho c)_w}\,\frac{r_1(r_1+\alpha+\beta)}{(r_1-r_3)(r_1-r_4)(r_1-r_5)(r_1-r_6)}\right] \\
& + \frac{1}{p-r_2}\left[\frac{\alpha D}{(\rho c)_w}\,\frac{r_2(r_2+\alpha+\beta)}{(r_2-r_3)(r_2-r_4)(r_2-r_5)(r_2-r_6)}\right] \\
& + \frac{1}{p-r_3}\left[\frac{\alpha}{(\rho c)_w}\,\frac{r_3(r_3+\alpha+\beta)}{(r_3-r_4)(r_3-r_5)(r_3-r_6)}\right]\left[\frac{C}{r_3-r_1}+\frac{D}{r_3-r_2}\right] \\
& + \frac{1}{p-r_4}\left[\frac{\alpha}{(\rho c)_w}\,\frac{r_4(r_4+\alpha+\beta)}{(r_4-r_3)(r_4-r_5)(r_4-r_6)}\right]\left[\frac{C}{r_4-r_1}+\frac{D}{r_4-r_2}\right] \\
& + \frac{1}{p-r_5}\left[\frac{\alpha}{(\rho c)_w}\,\frac{r_5(r_5+\alpha+\beta)}{(r_5-r_3)(r_5-r_4)(r_5-r_6)}\right]\left[\frac{C}{r_5-r_1}+\frac{D}{r_5-r_2}\right] \\
& + \frac{1}{p-r_6}\left[\frac{\alpha}{(\rho c)_w}\,\frac{r_6(r_6+\alpha+\beta)}{(r_6-r_3)(r_6-r_4)(r_6-r_5)}\right]\left[\frac{C}{r_6-r_1}+\frac{D}{r_6-r_2}\right]
\end{aligned} \qquad (34)$$

EQUATION 34 is of the form

$$\frac{\mu\eta}{\eta^2+(\frac{B}{L})^2} = \sum_{j=1}^{6}\frac{k_j}{p-r_j} \qquad (35)$$

*Descriptions of systems containing spatially uniform sources that vary linearly or sinusoidally in time are not deducible from the general solution of the present paper. Those cases are planned for analysis by Clark, Arpaci, and Treadwell,[2b] (II: Introduction), who also intend to solve the case of a spatially uniform source varying exponentially in time ($B = D = 0$ in EQUATION *a* above) for initial conditions that differ from those postulated in the present study.

in which the k_j terms are the bracketed constant coefficients in 34. The inverse transform of $k/(p-r)$ is ke^{rT}, so that

$$\frac{\mu\eta \sin (A - B\frac{x}{L})}{\eta^2 + (\frac{B}{L})^2} = \mathcal{L} \left\{ \sin (A - B\frac{x}{L}) \sum_{j=1}^{6} k_j e^{r_j T} \right\} \quad (36)$$

Similarly, the simpler second term in T_2 is expressible as

$$\frac{\mu (\frac{B}{L}) \cos (A - B\frac{x}{L})}{\eta^2 + (\frac{B}{L})^2} = \cos (A - B\frac{x}{L}) \sum_{j=1}^{6} \frac{n_j}{p - r_j} \quad (37)$$

in which

$$\begin{aligned}
n_1 &\equiv \left[\frac{aC}{(\rho c)_w} \frac{Bv}{L} \frac{r_1 + \beta}{(r_1 - r_3)(r_1 - r_4)(r_1 - r_5)(r_1 - r_6)} \right] \\
n_2 &\equiv \left[\frac{aD}{(\rho c)_w} \frac{Bv}{L} \frac{r_2 + \beta}{(r_2 - r_3)(r_2 - r_4)(r_2 - r_5)(r_2 - r_6)} \right] \\
n_3 &\equiv \left[\frac{a}{(\rho c)_w} \frac{Bv}{L} \frac{r_3 + \beta}{(r_3 - r_4)(r_3 - r_5)(r_3 - r_6)} \right] \left[\frac{C}{r_3 - r_1} + \frac{D}{r_3 - r_2} \right] \\
n_4 &\equiv \left[\frac{a}{(\rho c)_w} \frac{Bv}{L} \frac{r_4 + \beta}{(r_4 - r_3)(r_4 - r_5)(r_4 - r_6)} \right] \left[\frac{C}{r_4 - r_1} + \frac{D}{r_4 - r_2} \right] \\
n_5 &\equiv \left[\frac{a}{(\rho c)_w} \frac{Bv}{L} \frac{r_5 + \beta}{(r_5 - r_3)(r_5 - r_4)(r_5 - r_6)} \right] \left[\frac{C}{r_5 - r_1} + \frac{D}{r_5 - r_2} \right] \\
n_6 &\equiv \left[\frac{a}{(\rho c)_w} \frac{Bv}{L} \frac{r_6 + \beta}{(r_6 - r_3)(r_6 - r_4)(r_6 - r_5)} \right] \left[\frac{C}{r_6 - r_1} + \frac{D}{r_6 - r_2} \right]
\end{aligned} \quad (38)$$

Then

$$\frac{\mu (\frac{B}{L}) \cos (A - B\frac{x}{L})}{\eta^2 + (\frac{B}{L})^2} = \mathcal{L} \left\{ \cos (A - B\frac{x}{L}) \sum_{j=1}^{6} n_j e^{r_j T} \right\} \quad (39)$$

The complete inverse of $\overline{T}_2$ is the sum of the inverses in 36 and 39; thus

$$\left.\begin{aligned}
T_2 &= \sin (A - B\frac{x}{L}) \sum_{j=1}^{6} k_j e^{r_j T} + \cos (A - B\frac{x}{L}) \sum_{j=1}^{6} n_j e^{r_j T} \\
&= \sum_{j=1}^{6} \left[k_j \sin (A - B\frac{x}{L}) + n_j \cos (A - B\frac{x}{L}) \right] e^{r_j T}
\end{aligned}\right\} \quad (40)$$

which is valid for all r.

(3) *Inverse of* T_3. To invert T_3, it is necessary to invert (see EQUATIONS 27, 29, 35, 37),

$$-\left[\frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] e^{-\eta x}$$
$$= -\sum_{j=1}^{5} (k_j \sin A + n_j \cos A)\ \frac{e^{-\frac{px}{v}}\ (1+\frac{\alpha}{p+\beta})}{p - r_j} \tag{41}$$

Manipulating the exponent in the same fashion as in EQUATION 29, multiplying 41 by $(p + \beta)/(p + \beta)$, and rearranging $(p + \beta)/(p - r_j)$ as $[1 + (r_j + \beta)/(p - r_j)]$,

$$\frac{e^{-\frac{px}{v}}\ (1+\frac{\alpha}{p+\beta})}{p - r_j} = \frac{e^{-\frac{\alpha x}{v}}\ e^{-\frac{px}{v}}\ e^{\frac{\alpha\beta x/v}{p+\beta}}}{p+\beta} + \frac{(r_j+\beta)\,e^{-\frac{\alpha x}{v}}\ e^{-\frac{px}{v}}\ e^{\frac{\alpha\beta x/v}{p+\beta}}}{(p-r_j)\,(p+\beta)} \tag{42}$$

The first term on the right is identical with the first term in 30 and has the same inverse (EQUATION 31). Thus, it will give rise in T_3 to the terms

$$-H(\phi)e^{-\frac{\alpha x}{v}} \sum_{j=1}^{6} \left[k_j \sin A + n_j \cos A\right] e^{-\beta\phi}\ I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) \tag{43}$$

in which the unit step function $H(\phi)$ again signifies that 43 applies only for $\phi = (r - \frac{x}{v}) > 0$.

To invert the second term in 42, that term is treated as the product of two transforms. Using

$$\frac{1}{p - r_j} = \mathcal{L}\left\{e^{r_j \tau}\right\} \equiv \mathcal{L}\left\{F_1(\tau)\right\} \tag{44}$$

and

$$\frac{e^{-\frac{px}{v}}\ e^{\frac{\alpha\beta x/v}{p+\beta}}}{p+\beta} = \mathcal{L}\left\{H(\phi)e^{-\beta\phi} I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right)\right\} \equiv \mathcal{L}\left\{F_2(\tau)\right\} \tag{45}$$

it follows that

$$\frac{1}{p - r_j}\left(\frac{e^{-\frac{px}{v}}\ e^{\frac{\alpha\beta x/v}{p+\beta}}}{p+\beta}\right) = \mathcal{L}\{F_1(\tau)\}\ \mathcal{L}\{F_2(\tau)\} \tag{46}$$

and by the Superposition theorem (Carslaw and Jaeger,[1] p. 243),

$$\mathcal{L}\{F_1(\tau)\}\ \mathcal{L}\{F_2(\tau)\} = \mathcal{L}\left\{\int_0^\tau F_2(\xi)\,F_1(\tau-\xi)\,d\xi\right\}$$
$$= \mathcal{L}\left\{\int_0^\tau H(\xi-\tfrac{x}{v})\,e^{-\beta(\xi-\frac{x}{v})}\,I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta(\xi-\tfrac{x}{v})}\right)\,e^{r_j(\tau-\xi)}\,d\xi\right\} \quad (47)$$

in which the definitions of $F_1(\tau)$ and $F_2(\tau)$ as given in 44 and 45 have been used. It is noted that the presence of $H(\xi-\frac{x}{v})$ under the integral signifies that 47 applies only for $(\xi-\frac{x}{v})>0$.

The argument of integration in 47 is now changed to $\sigma \equiv (\xi-\frac{x}{v})$. The upper limit of the integrand becomes $(\tau-\frac{x}{v})$ which again can be denoted by ϕ, and while the lower limit formally becomes $-\frac{x}{v}$, values of σ in the range $-\frac{x}{v} \le \sigma \le 0$ cannot contribute to the solution as then $H(\sigma)=0$.

The exponent $r_j(\tau-\xi)$ on e in the integrand is written

$$r_j(\tau-\xi) \equiv r_j\left[(\tau-\tfrac{x}{v})-(\xi-\tfrac{x}{v})\right] \equiv r_j\left[(\tau-\tfrac{x}{v})-\sigma\right]$$

and it is noted that $(\tau - x/v)$, being free of σ, can be removed from under the integral sign and be denoted by ϕ.

The unit step function $H(\sigma)$ shows that the integral can contribute only for positive values of the argument. Thus the upper limit, $\phi = (\tau-\frac{x}{v})$, must also be positive; that is, EQUATION 48 applies only for $(\tau-\frac{x}{v})>0$. Further, since the step function $H(\sigma)$ will always equal unity in the positive range $0<\sigma\le(\tau-\frac{x}{v})$, that function may be removed from under the integral sign and be replaced by $H(\phi)$ there. Thus the second term on the right in 42 gives rise in T_3 to the terms,

As a result of these steps, 46 and 47 together become

$$\frac{1}{p-r_j}\left(\frac{e^{-\frac{px}{v}}e^{\frac{\alpha\beta x/v}{p+\beta}}}{p+\beta}\right) = \mathcal{L}\left\{e^{r_j\phi}\int_0^\phi H(\sigma)\,e^{-(r_j+\beta)\sigma}\,I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right)d\sigma\right\} \quad (48)$$

$$-H(\phi)e^{-\frac{\alpha x}{v}}\sum_{j=1}^{6}(k_j\sin A + n_j\cos A)(r_j+\beta)\,e^{r_j\phi}$$
$$\int_0^\phi e^{-(r_j+\beta)\sigma}\,I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right)d\sigma \quad (49)$$

Combining 43 and 49 to obtain the complete inverse of $\bar{T}_3$,

$$T_3 = -H(\phi)\, e^{-\frac{\alpha x}{v}} \sum_{j=1}^{6} \left(k_j \sin A + n_j \cos A \right) \left[e^{-\beta\phi}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) \right. \tag{50}$$

$$\left. + (r_j + \beta)\, e^{r_j \phi} \int_o^{\phi} e^{-(r_j+\beta)\sigma}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right) d\sigma \right]$$

A simplification of 50 is possible if each integral-containing term is integrated once by parts. This procedure eliminates the bracketed non-integral-containing term in 50 and removes the coefficient $(r_j + \beta)$ of the integral-containing term. The integrand then involves the modified Bessel function of first order, I_1, and a denominator term $\sigma^{1/2}$.

(4) *Formula for fluid temperature.* Using 28, 31, 40 and 50, rearranging slightly, and replacing the argument ψ in 31 by the symbol σ,

$$T = \left\{ t_o + \sum_{j=1}^{6} \left[k_j \sin\left(A - B\tfrac{x}{L}\right) + n_j \cos\left(A - B\tfrac{x}{L}\right) \right] e^{r_j \tau} \right\}$$

$$+ H(\phi)\, e^{-\frac{\alpha x}{v}} \left\{ (T_o - t_o) \left[e^{-\beta\phi}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) + \beta \int_o^{\phi} e^{-\beta\sigma} I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right) d\sigma \right] \right. \tag{51}$$

$$- \sum_{j=1}^{6} (k_j \sin A + n_j \cos A) \left[e^{-\beta\phi} I_o \left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) \right.$$

$$\left. \left. + (r_j + \beta)\, e^{r_j \phi} \int_o^{\phi} e^{-(r_j+\beta)\sigma}\, I_o \left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right) d\sigma \right] \right\}$$

Solution for Wall Temperature

The transform of the wall temperature is obtained by substituting 24 into 16. Thus

$$\bar{t} = \bar{t}_1 + \bar{t}_2 + \bar{t}_3 + \bar{t}_4 \tag{52}$$

where

$$\bar{t}_1 \equiv \frac{t_o}{p+\beta} + \frac{\beta}{p+\beta} \left[\frac{t_o}{p} + \left(\frac{T_o - t_o}{p} \right) e^{-\eta x} \right] \tag{53}$$

$$\bar{t}_2 \equiv \frac{\beta}{p+\beta} \left[\frac{\mu\eta \sin\left(A - B\tfrac{x}{L}\right) + \mu\left(\tfrac{B}{L}\right) \cos\left(A - B\tfrac{x}{L}\right)}{\eta^2 + \left(\tfrac{B}{L}\right)^2} \right] \tag{54}$$

$$\bar{t}_3 \equiv -\frac{\beta}{p+\beta}\left[\frac{\mu\eta \sin A + \mu\left(\frac{B}{L}\right)\cos A}{\eta^2 + \left(\frac{B}{L}\right)^2}\right] e^{-\eta x} \tag{55}$$

$$\bar{t}_4 \equiv \frac{\sin\left(A - B\frac{x}{L}\right)}{(\rho c)_w}\left[\frac{1}{p+\beta}\left(\frac{C}{p-r_1} + \frac{D}{p-r_2}\right)\right] \tag{56}$$

$\bar{t}_1$ is the inverse of the wall temperature in a source-free system and is given by Carslaw and Jaeger[1] (EQUATION 15, p. 328). In the present notation,

$$t_1 = t_o + \beta\,(T_o - t_o)\, e^{-\frac{\alpha x}{v}} \int_o^{\phi} H(\sigma)\, e^{-\beta\sigma}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\,\beta\sigma}\right) d\sigma \tag{57}$$

which applies only when $\phi > 0$.

To invert $\bar{t}_2$, it is written explicitly in terms of p, using 35 and 37.

$$\bar{t}_2 = \beta \sum_{j=1}^{6} \frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{(p+\beta)(p-r_j)} \tag{58}$$

which breaks into partial fractions as

$$\bar{t}_2 = \beta \sum_{j=1}^{6} \frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{r_j + \beta}\left(\frac{1}{p-r_j} - \frac{1}{p+\beta}\right) \tag{59}$$

and inverts directly to

$$t_2 = \beta \sum_{j=1}^{6} \frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{r_j + \beta}\left(e^{r_j \tau} - e^{-\beta\tau}\right) \tag{60}$$

To invert $\bar{t}_3$, 41 is used and $\bar{t}_3$ is written

$$\bar{t}_3 = -\beta \sum_{j=1}^{6} (k_j \sin A + n_j \cos A)\, \frac{e^{-\frac{px}{v}}\left(1 + \frac{\beta}{p+\beta}\right)}{(p+\beta)(p-r_j)} \tag{61}$$

and on comparison with 48, inverts to

$$t_3 = -H(\phi)\,\beta\, e^{-\frac{\alpha x}{v}} \sum_{j=1}^{6} (k_j \sin A + n_j \cos A)$$
$$e^{r_j \phi} \int_o^{\phi} e^{-(r_j + \beta)\sigma}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\,\beta\sigma}\right) d\sigma \tag{62}$$

Finally, $\bar{t}_4$ is broken into partial fractions and inverts to

$$t_4 = \frac{\sin\left(A - B\frac{x}{L}\right)}{(\rho c)_w}\left[\frac{C}{r_1+\beta}\left(e^{r_1\tau} - e^{-\beta\tau}\right) + \frac{D}{r_2+\beta}\left(e^{r_2\tau} - e^{-\beta\tau}\right)\right] \quad (63)$$

The complete solution for wall temperature is the sum of the expressions in 57, 60, 62 and 63. Rearranging slightly,

$$\begin{aligned} t = t_o &+ \frac{\sin\left(A - B\frac{x}{L}\right)}{(\rho c)_w}\left[\frac{Ce^{r_1\tau}}{r_1+\beta} + \frac{De^{r_2\tau}}{r_2+\beta}\right] \\ &+ \beta\sum_{j=1}^{6}\left[\frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{r_j+\beta}\right] e^{r_j\tau} \\ &+ H(\phi)\, e^{-\frac{ax}{v}}\left[(T_o - t_o)\ \beta \int_o^{\phi} e^{-\beta\sigma}\, I_o\left(2\sqrt{\tfrac{ax}{v}\beta\sigma}\right)\, d\sigma\right. \\ &\left. - \sum_{j=1}^{6}(k_j \sin A + n_j \cos A)\,\beta e^{r_j\phi}\int_o^{\phi} e^{-(r_j+\beta)\sigma} I_o\left(2\sqrt{\tfrac{ax}{v}\beta\sigma}\right)\, d\sigma\right] \\ &- \frac{\sin\left(A - B\frac{x}{L}\right)}{(\rho c)_w}\left[\frac{C}{r_1+\beta} + \frac{D}{r_2+\beta}\right] e^{-\beta\tau} \\ &- \beta\sum_{j=1}^{6}\left[\frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{r_j+\beta}\right] e^{-\beta\tau} \end{aligned} \quad (64)$$

It is now shown that the last two expressions vanish, that is, the complete coefficient of $e^{-\beta\tau}$ is equal to zero. The terms involving $\cos\left(A - B\frac{x}{L}\right)$ are considered first; it is required to show that

$$\sum_{j=1}^{6}\frac{n_j}{r_j+\beta} = 0 \quad (65)$$

From the definitions of n_j, the explicit form of 65 (neglecting the term $aBv/L(\rho c)_w$, common to every n), is

$$\begin{aligned} &\frac{C}{(r_1-r_3)(r_1-r_4)(r_1-r_5)(r_1-r_6)} + \frac{D}{(r_2-r_3)(r_2-r_4)(r_2-r_5)(r_2-r_6)} \\ &+ \frac{C}{(r_3-r_1)(r_3-r_4)(r_3-r_5)(r_3-r_6)} + \frac{D}{(r_3-r_2)(r_3-r_4)(r_3-r_5)(r_3-r_6)} \end{aligned} \quad (66)$$

$$+\frac{C}{(r_4-r_1)(r_4-r_3)(r_4-r_5)(r_4-r_6)}+\frac{D}{(r_4-r_2)(r_4-r_3)(r_4-r_5)(r_4-r_6)} \quad (66)$$

$$+\frac{C}{(r_5-r_1)(r_5-r_3)(r_5-r_4)(r_5-r_6)}+\frac{D}{(r_5-r_2)(r_5-r_3)(r_5-r_4)(r_5-r_6)}$$

$$+\frac{C}{(r_6-r_1)(r_6-r_3)(r_6-r_4)(r_6-r_5)}+\frac{D}{(r_6-r_2)(r_6-r_3)(r_6-r_4)(r_6-r_5)}$$

The first term in 66 is now resolved into partial fractions:

$$\frac{C}{(r_1-r_3)(r_1-r_4)(r_1-r_5)(r_1-r_6)}=\frac{C}{(r_1-r_3)}\left[\frac{1}{(r_3-r_4)(r_3-r_5)(r_3-r_6)}\right]$$

$$+\frac{C}{(r_1-r_4)}\left[\frac{1}{(r_4-r_3)(r_4-r_5)(r_4-r_6)}\right]+\frac{C}{(r_1-r_5)}\left[\frac{1}{(r_5-r_3)(r_5-r_4)(r_5-r_6)}\right] \quad (67)$$

$$+\frac{C}{(r_1-r_6)}\left[\frac{1}{(r_6-r_3)(r_6-r_4)(r_6-r_5)}\right]$$

each term of which is the negative of, and therefore cancels, an analogous remaining term involving C in 66. In exactly the same way it is shown that the terms involving D in 66 also vanish. Thus the validity of 65 is demonstrated.

The terms involving $\sin\left(A - B\frac{x}{L}\right)$ are now considered. From 64, it is required to show that

$$\left[\frac{C}{(\rho c)_w}\frac{1}{r_1+\beta}+\frac{\beta k_1}{r_1+\beta}+\frac{D}{(\rho c)_w}\quad\frac{1}{r_2+\beta}+\frac{\beta k_2}{r_2+\beta}+\sum_{j=3}^{6}\frac{\beta k_j}{r_j+\beta}\right]=0 \quad (68)$$

The term $(\beta k_1/r_1+\beta)$ is written explicitly using the definition of k_1,

$$\frac{\beta k_1}{r_1+\beta}\equiv\frac{a\beta C}{(\rho c)_w}\quad\frac{r_1(r_1+a+\beta)}{(r_1+\beta)(r_1-r_3)(r_1-r_4)(r_1-r_5)(r_1-r_6)} \quad (69)$$

The right member of 69 resolves into partial fractions,

$$\frac{a\beta C}{(\rho c)_w}\left[\frac{1}{r_1-r_3}\frac{r_3(r_3+a+\beta)}{(r_3+\beta)(r_3-r_4)(r_3-r_5)(r_3-r_6)}+\frac{1}{r_1-r_4}\frac{r_4(r_4+a+\beta)}{(r_4+\beta)(r_4-r_3)(r_4-r_5)(r_4-r_6)}\right.$$

$$\left.+\frac{1}{r_1-r_5}\frac{r_5(r_5+a+\beta)}{(r_5+\beta)(r_5-r_3)(r_5-r_4)(r_5-r_6)}+\frac{1}{r_1-r_6}\frac{r_6(r_6+a+\beta)}{(r_6+\beta)(r_6-r_3)(r_6-r_4)(r_6-r_5)}\right]$$

$$+\frac{a\beta C}{(\rho c)_w}\quad\frac{1}{r_1+\beta}\quad\frac{-\beta(-\beta+a+\beta)}{(-\beta-r_3)(-\beta-r_4)(-\beta-r_5)(-\beta-r_6)} \quad (70)$$

Comparing the bracketed terms in 70 with the analogous terms involving C in the expression

$$\sum_{j=3}^{6} \frac{\beta k_j}{r_j + \beta}$$

it is seen that the analogous terms have the same absolute values, but opposite signs. Hence in 68, the terms involving C reduce to the sum

$$\frac{C}{(\rho c)_w} \frac{1}{r_1+\beta} + \frac{a\beta C}{(\rho c)_w} \frac{1}{r_1+\beta} \quad \frac{-a\beta}{(-\beta-r_3)(-\beta-r_4)(-\beta-r_5)(-\beta-r_6)}$$

or

$$\frac{C}{(\rho c)_w} \frac{1}{r_1+\beta} \left[1 - \frac{a^2\beta^2}{(-\beta-r_3)(-\beta-r_4)(-\beta-r_5)(-\beta-r_6)}\right] \qquad (71)$$

The denominator in the bracket of 71, if multiplied out, has the value that would be obtained on replacing p by $-\beta$ in the intermediate member of EQUATION 33; that is, its value is

$$(-\beta)^4 + (-\beta)^3 [2a + 2\beta] + (-\beta)^2 \left[(a+\beta)^2 + \left(\frac{Bv}{L}\right)^2\right]$$
$$+ (-\beta) \left[2\beta \left(\frac{Bv}{L}\right)^2\right] + \beta^2 \left(\frac{Bv}{L}\right)^2$$

which reduces to $a^2\beta^2$, so that the bracketed term of 71 is equal to zero. In exactly the same fashion it follows that the terms involving D in EQUATION 68 also vanish.

Accordingly the complete coefficient of $e^{-\beta\tau}$ in 64 is zero, and the final expression for the wall temperature is

$$t = t_o + \frac{\sin\left(A - \beta\frac{x}{L}\right)}{(\rho c)_w} \left[\frac{Ce^{r_1\tau}}{r_1+\beta} + \frac{De^{r_2\tau}}{r_2+\beta}\right]$$
$$+ \beta \sum_{j=1}^{6} \left[\frac{k_j \sin\left(A - B\frac{x}{L}\right) + n_j \cos\left(A - B\frac{x}{L}\right)}{r_j + \beta}\right] e^{r_j\tau}$$
$$+ H(\phi)\, e^{-\frac{\alpha x}{v}} \left[(T_o - t_o)\beta \int_o^{\phi} e^{-\beta\sigma} I_o\left(2\sqrt{\frac{\alpha x}{v}\beta\sigma}\right) d\sigma\right. \qquad (72)$$
$$\left. - \sum_{j=1}^{6} (k_j \sin A + n_j \cos A)\beta e^{r_j\phi} \int_o^{\phi} e^{-(r_j+\beta)\sigma} I_o\left(2\sqrt{\frac{\alpha x}{v}\beta\sigma}\right) d\sigma\right]$$

Attention is now turned to a simplification possible in the solution for T (EQUATION 51). By resolving any one k_j and any one n_j into their

respective partial fractions, in a fashion similar to that employed in EQUATION 67, it is seen that

$$\sum_{j=1}^{6} k_j = \sum_{j=1}^{6} n_j = 0 \tag{73}$$

Accordingly in 51,

$$-H(\phi)\, e^{-\frac{\alpha x}{v}} \sum_{j=1}^{6} (k_j \sin A + n_j \cos A)\, e^{-\beta\phi}\, I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) = 0 \tag{74}$$

Then 51 reduces to

$$T = t_o + \sum_{j=1}^{6} \left[k_j \sin\left(A - B\tfrac{x}{L}\right) + n_j \cos\left(A - B\tfrac{x}{L}\right)\right] e^{r_j \tau}$$

$$+H(\phi)\, e^{-\alpha x/v}\left\{(T_o - t_o)\left[e^{-\beta\phi} I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\phi}\right) + \beta \int_o^{\phi} e^{-\beta\sigma} I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right) d\sigma\right]\right. \tag{75}$$

$$\left. -\sum_{j=1}^{6} (k_j \sin A + n_j \cos A)(r_j + \beta)\, e^{r_j \phi} \int_o^{\phi} e^{-(r_j+\beta)\sigma} I_o\left(2\sqrt{\tfrac{\alpha x}{v}\beta\sigma}\right) d\sigma\right\}$$

which is the final equation for the fluid temperature.

Validity of Results

EQUATIONS 72 and 75 for t *and* T satisfy differential EQUATIONS 1 and 4, as may be checked by insertion of the solutions for t and T into those differential equations. EQUATION 1 is found to be satisfied straightforwardly. In demonstrating that EQUATION 4 is satisfied, use is made of the relation between k_j and n_j, found from their definitions in 34 and 38; and the fact is used that if an r_j ($j = 3, 4, 5, 6$) is substituted for p in the left member of 33, the resulting expression equals zero.

EQUATION 72 satisfies the initial condition on the wall temperature, EQUATION 2. For at $\tau = 0$, ϕ is negative, $H(\phi)$ is equal to zero, and the integral-containing terms disappear. Also, since $e^{r_j \tau}$ equals unity, the resulting total coefficients of cos $(A - B\frac{x}{L})$ and sin $(A - B\frac{x}{L})$ respectively vanish (EQUATIONS 65 to 71). Thus, at the start of the transient, $t = t_o$, as required.

EQUATION 75 satisfies the initial and boundary conditions shown in EQUATIONS 5 to 7. At $\tau = 0 = H(\phi)$, the expression multiplied by $H(\phi)$ vanishes. Since $e^{r_j \tau} = 1$, the summation term also disappears (EQUATION 73). Hence, $T = T_o$ as required by EQUATION 5. On the other hand, when $x = 0$, $\phi = \tau$ and $H(\phi) = 1$. Recalling that $I_o(0) = 1$, the integrals in the expression multiplied by $H(\phi)$ are easily evaluated and, using

EQUATION 73, it is found that this expression reduces to

$$1 \cdot e^{-\frac{ax}{v}} [(T_o - t_o) - \sum_{1}^{6} (k_j \sin A + n_j \cos A) e^{rj\tau}]$$

Since when $x = 0$, $e^{-\frac{ax}{v}} = 1$, it follows that the summation terms in 75 cancel one another, the t_o disappears, and $T = T_o$ as required by EQUATIONS 6 and 7.

Since EQUATIONS 72 and 75 satisfy the differential equations, initial and boundary conditions, they are considered valid solutions for wall and fluid temperatures in the present system.

COMMENTS

The fluid and wall temperatures consist of two types of terms, the first valid for all τ, the second valid only for $\tau > x/v$.

The first type of term reflects the fact that at every point the heat source exerts an immediate and continuous local effect on both the fluid and wall temperatures. In the present system, this effect is exponential in time; a function of the spatial distribution of source strength and of its first derivative appears as a coefficient multiplying the exponential function of time.

The second type of term reflects the contributions of all the points of the channel upstream of x to the fluid and wall temperatures at the point x. This term applies only after the fluid, entering the channel with velocity v, has had an opportunity to reach the point x.

EQUATIONS 72 and 75 permit calculation of the proper combination of reactor power, fluid flow rate, and inlet temperature required to avoid hot spots and thermal stresses in the wall and boiling in the fluid, and provide information toward calculation of the nuclear reactivity as affected by reactor material temperatures.

Numerical values useful for the warm-up period (prior to the start of heat generation), are presented in FIGURES 1 and 2 that apply to source-free channels.

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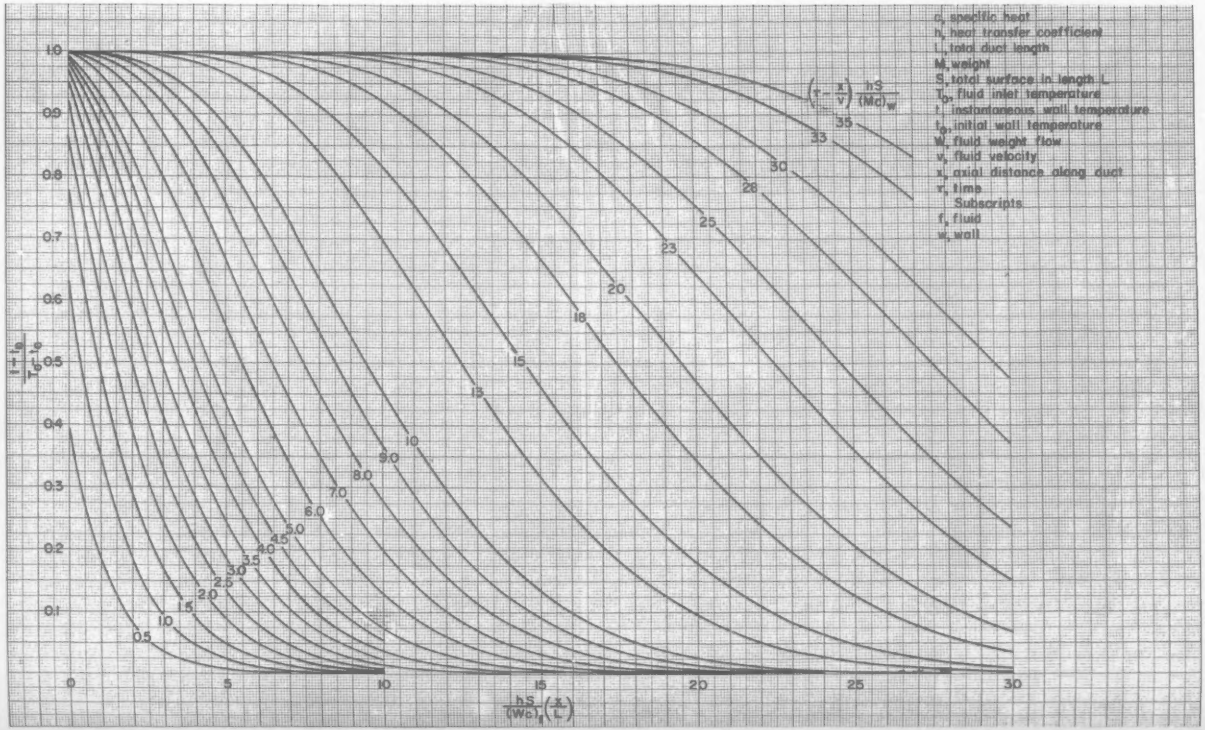

FIGURE 1. Wall temperature versus time and position in source-free system.

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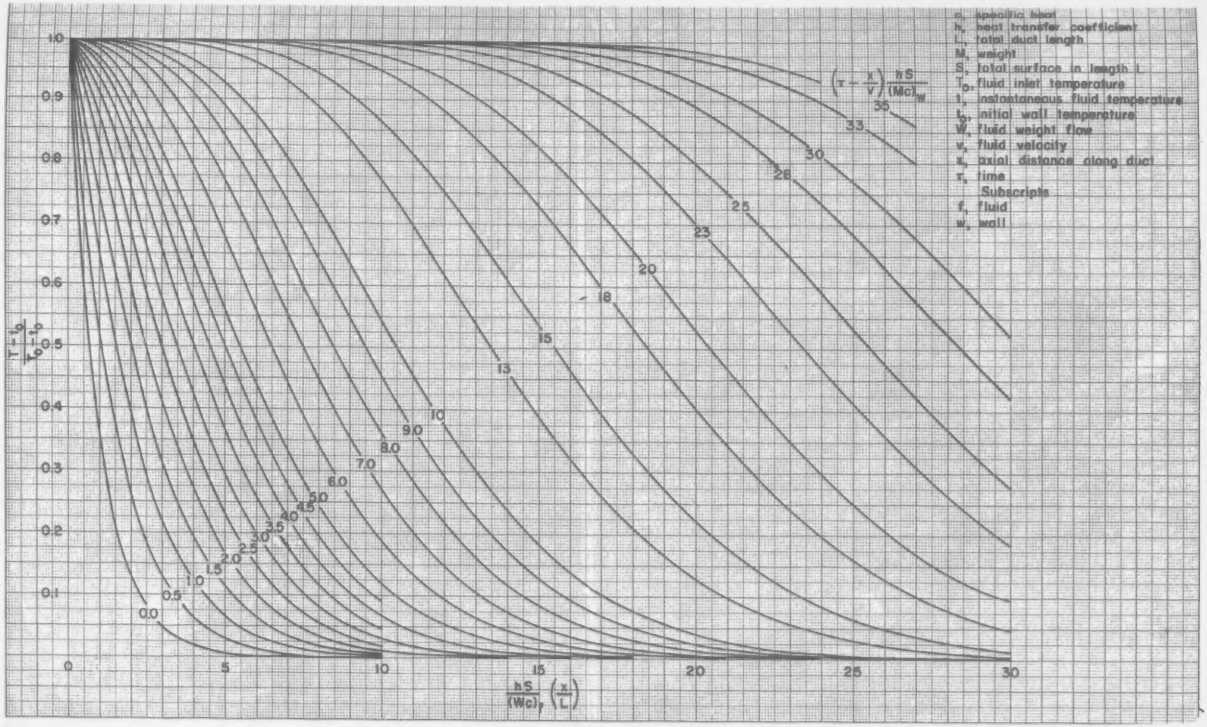

FIGURE 2. Fluid temperature versus time and position in source-free system.

SECTION OF PSYCHOLOGY

VISUAL IMAGES: "INDUCED HALLUCINATIONS"*

By Walter S. Boernstein

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The experiments reported here deal mainly with the mutual relationship between sensory processes and body tonus in man. Body tonus may be defined as the dynamic muscular balance that guarantees the body's appropriate posture and attitudes in adjusting to the varying conditions of life. This feat is achieved by involuntary tensing and relaxing of the muscles in repose or movement. The entire nervous system is involved in this task, but chiefly such areas as certain parts of the frontal lobes and cerebellum, the basal ganglia, and the reticular system.

I showed that the body tonus is involved in perceptions of all kinds when, stimulated by statements made by H. von Helmholtz [1] and E. Mach, [2] I developed the *theory of specific sensory-motor synergy*. [3a,b] This theory holds that "for a sensory stimulus to become effective, a twofold function must take place – centripetal and centrifugal ..." [4]

Many authors have since agreed that changes in the body tonus influence perception: among others, Heinz Werner [5] who, incidentally, later also advanced a "sensory-tonic field theory of perception." Moreover, it was shown by J. Loeb, [6] E. Metzger, [7] and others that light stimuli, acting through the eye, influence the body tonus. Metzger in particular proved that short-wave light – for example, blue – increases the body tonus, and that long-wave light – for example, red – decreases it, amplitudes and other conditions being equal.

The sense of vision fulfills at least two functions: (1) it engenders a specific type of perceptual process – that of seeing the world outside; and (2) it makes a powerful contribution toward regulating the body tonus – thereby alerting and stabilizing the organism. The first of these functions is mediated chiefly through the thalamocortical pathway, while the latter is subserved by other channels.

This dichotomy is not confined to vision. I observed the same effect on the body tonus by *acoustic* stimuli in a veteran with a cerebellar lesion. When this patient had his eyes closed, his outstretched arms involuntarily moved downward and outward. However, when I played a high tone on the E string of the violin, his arms rose slightly in a jerky way. On the other hand, when he heard a low tone played on the G string, his arms lowered in the same manner. Each time a tone faded away the arms tended to jerk back to the starting position. Therefore, it is clear that

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on October 21, 1957. The experimental material validating these concepts will be published later.

sounds, too, have an effect on the body tonus, the high tones acting like short-wave light, the low tones like long-wave light.

Two categories of response to optic and acoustic stimuli may be distinguished. The first is specific, a perception dictated by the modality of the sense; the other nonspecific, a response of the body tonus brought about by stimuli acting on either the visual or the acoustic sense. This latter effect is also perceived; it is an "a-modal" quality, bright or dark – adjectives applied to the acoustic field by C. Stumpf, F. von Brentano, and W. Köhler.[8]

E. M. von Hornbostel[9] showed that sensory stimuli of other fields also give rise to bright-dark perception. "Brightness," he wrote, "is a property common to all perceptual phenomena... identical in all senses... phylogenetically one of the oldest perceptual phenomena."

Accordingly, in a series of experiments I was able to demonstrate that bright nonoptic sensory stimuli, namely bright odors and high sounds, add to the optic brightness of a dimly lit opaque glass screen and vice versa.[10] Another series, limited in number, demonstrated that the same kinds of stimuli could influence the Purkinje phenomenon.[11] In further experiments, with subjects who were placed in a state of dark adaptation, I have been able to show that nonoptic sensory stimuli can produce visual images, the nature of which is determined by the degree of brightness of the stimulus.[12-14]

Positive results were obtained in 60 of 100 subjects. In these instances, bright odors, tastes, or sounds elicited bright, pleasant images; dark ones, the reverse. The visual images covered the whole range from indistinct, fleeting "entoptic" phenomena (phosphenes) to lifelike scenes in motion and color. In other cases, even mere radiation of the skin by ultraviolet or infrared rays caused vivid optic images.

From these experiments on man, as well as from special work on the melanophores of the frog,[15] it would seem that brightness is a quality that is, indeed, closely related to increase of body tonus. It is mediated through the pituitary gland.

Finally, it is suggested that strong tonus-increasing forces, such as violent emotions, especially when repressed, can provoke actual hallucinations in the same way that "induced hallucinations" are provoked in a state of dark adaptation.

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DIVISION OF MYCOLOGY

THE COMMUNICABLE DISEASE CENTER'S FILM TEACHING AIDS IN MEDICAL MYCOLOGY*

By Libero Ajello

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The full public health importance of human mycotic diseases has been realized only in recent years. Formerly, fungus infections of the skin and vital organs were considered either to be rare in occurrence or confined to the humid, tropical areas of the world. Today it is known that pathogenic fungi are prevalent in the United States and that they induce diseases that pose difficult diagnostic and therapeutic problems.

Of approximately 54 human pathogenic or potentially pathogenic fungus species, 38 occur naturally or have become established in the United States (TABLE 1). *Blastomyces brasiliensis* and *Nocardia brasiliensis* are the only major systemic pathogens not encountered.

Dermatophyte species such as *Trichophyton mentagrophytes, T. rubrum, T. tonsurans,* and *Microsporum audouinii* infect millions of individuals. Ringworm infections, although not usually disabling, cause considerable annoyance, present diagnostic problems, and lead to the expenditure of literally millions of dollars for the purchase of medications. Through drug trade channels, it has been determined that more than $25,000,000 was expended during 1956 by the American public for antimycotic drugs.

The Mississippi-Ohio River Valley area is the endemic center for *Histoplasma capsulatum.* One can conservatively estimate that 25 to 30,000,000 of that region's inhabitants have been infected by this fungus. Leo M. Furcolow† estimates that half a million people become infected yearly in the states of Tennessee, Kentucky, Missouri, Ohio, Kansas, Indiana, Arkansas, and Illinois, and that 800 deaths due to histoplasmosis occur annually. The public health importance of mycotic diseases is thus demonstrated by the number of humans infected, the economic burden on the population, and the high morbidity rates.

To the clinician, the mycoses pose difficult diagnostic problems. The clinical signs of the majority of fungus diseases are not clear-cut. Without the aid of specific medical mycologic tests such infections are frequently misdiagnosed.

With the increased interest in mycotic diseases there is a distinct need to orient and train physicians and laboratory workers in the clinical

*This paper, illustrated with a motion picture, was presented at a meeting of the Division of Mycology on October 25, 1957.

†Personal communication, Communicable Disease Center, Kansas City Field Station, Kansas City, Kans.

TABLE 1

SPECIES OF HUMAN PATHOGENIC FUNGI

Established within the United States	*Not established within the United States*
Absidia corymbifera	*Blastomyces brasiliensis*
A. ramosa	*Cephalosporium falciformis*
Actinomyces bovis	*C. recifei*
Allescheria boydii	*Cladosporium carrionii*
Aspergillus fumigatus	*Madurella mycetomi*
Blastomyces dermatitidis	*Microsporum distortum*
Candida albicans	*Nocardia brasiliensis*
C. guilliermondii	*Phialophora dermatitidis*
C. krusei	*Piedraia hortai*
Cladosporium trichoides	*Streptomyces somaliensis*
C. werneckii	*Trichophyton concentricum*
Coccidioides immitis	*T. ferrugineum*
Cryptococcus neoformans	*T. gallinae*
Epidermophyton floccosum	*T. megninii*
Geotrichum candidum	*T. soudanensis*
Histoplasma capsulatum	*Trichosporon cutaneum*
Madurella grisea	
Malassezia furfur	
Microsporum audouinii	
M. canis	
M. gypseum	
Nocardia asteroides	
Phialophora compactum	
P. jeanselmei	
P. pedrosoi	
P. verrucosum	
Rhinosporidium seeberii	
Rhizopus arrhizus	
R. oryzae	
Sporotrichum schenckii	
Streptomyces madurae	
S. pelletierii,	
Trichophyton equinum	
T. mentagrophytes	
T. rubrum	
T. schoenleinii	
T. tonsurans	
T. verrucosum	

aspects of mycotic diseases and the laboratory procedures employed in the detection, isolation, and identification of human pathogenic fungi.

Audio-visual aids of various types are useful tools to those concerned with medical mycologic instruction. They are best used to supplement lectures. Clinical cases of various mycotic diseases, even in medical centers, are not always available for demonstration purposes when desired. An adequate collection of color photographs of lesions, tissue

sections, and the gross and microscopic features of the etiological agents requires much time and effort to accumulate. Thus it was felt by the staff of the Medical Mycology Unit of the Communicable Disease Center that the preparation and distribution of film strips, slide series, and motion pictures on mycologic subjects would prove to be a valuable service to those engaged in teaching medical mycology. Since 1950 eight film strips, two slide series, and a motion picture have been prepared by our unit together with the Audio-Visual Section of the Communicable Disease Center.

Film strips. (1) *Laboratory Diagnosis of Tinea Capitis in Children (Microsporum Infections),* 5-127-0; (2) *A Mycological Slide Culture Technique,* F 24; (3) *Laboratory Diagnosis of Trichophyton Infections: Part I, Ectothrix Infections of the Beard and Scalp,* 94 A; (4) *Laboratory Diagnosis of Trichophyton Infections: Part II, Endothrix Infections of the Scalp,* 94 B; (5) *Laboratory Identification of Coccidioides Immitis,* F 116B; (6) *Laboratory Identification of Blastomyces Dermatitidis,* F 116 A; (7) *Laboratory Diagnosis of Ringworm in Animals: Part I, Microsporum Infections,* F 221; and (8) *Laboratory Diagnosis of Ringworm in Animals: Part II, Trichophyton Infections,* F 22 A.

These film strips are composed of an integrated series of color slides that deal with specific laboratory procedures. They describe the detection of pathogenic fungi in clinical materials and the methods used for their isolation and identification. In each strip the clinical manifestations of the disease caused by the fungi are fully illustrated in order to guide and orient the student of medical mycology.

In addition to the printed text, a recorded narration accompanies each film strip. The instructor may make use of this material or prepare comments according to specific needs.

One strip – *A Mycological Slide Culture Technique* – is concerned solely with a pictorial description of one of the most useful methods for observing the undisturbed relationship of spores and conidiophores. This type of information is frequently vital for the identification of certain fungi.

Slide series (2 x 2 slides). (1) *Histopathology of Human Mycoses* (108 slides), F 13 A,B,C,D : *Part I, Superficial Fungus Infections* (15 slides), F 13 A; *Part II, Cutaneous Fungus Infections* (16 slides), F 13 B; *Part III, Subcutaneous Fungus Infections* (17 slides), F 13 C; and *Part IV, Systemic Fungus Infections* (60 slides), F 13 D. The appearance of human pathogenic fungi in tissues and host tissue reactions are presented in this group of slides. The majority of the etiological agents in the four major categories of the mycoses are covered.

(2) *Common Saprophytic Fungi* (173 slides), S 170. Nonpathogenic fungi are frequently encountered as contaminants in the course of laboratory work in medical mycology. Acquaintanceship with the more common of of the saprophytic molds facilitates the work of the medical mycologist.

Contaminants thus can be quickly screened and eliminated from further consideration and full attention paid to the pathogens.

Motion picture. (1) *Coccidioidomycosis: Its Epidemiological and Clinical Aspects* (20 min.). Color, 16mm., sound. 237. The motion picture on coccidioidomycosis is presented as an example of the type of material that has been prepared. The film was made in cooperation with Roger Egeberg, then of the Veterans Administration Center in Los Angeles, Calif., and now Medical Director of the Los Angeles County General Hospital, also in Los Angeles.

Coccidioidomycosis is one of the most important of human diseases caused by molds. In the United States several million people have been infected by the fungus *Coccidioides immitis,* which causes the disease, and each year several thousand new infections occur among the inhabitants and visitors in certain areas in the southwestern states of Arizona, California, Nevada, New Mexico, Texas, and Utah. The disease is also known to occur in Central and South America, but it does not occur in Europe, Asia, Africa, or Australia.

Because *C. immitis* elicits a wide variety of symptoms in susceptible individuals, diagnosis is not simple. Most infections originate in the lungs, as the spores of the fungus are blown about by winds and are inhaled. Patients develop signs of respiratory diseases such as those of a cold, influenza, or pneumonia. They may have fever, night sweats, chest pains, and malaise. X-ray examination, skin tests, and serologic tests, together with demonstration of the fungus in tissue and its cultural isolation, are of great aid to the physician in making a correct diagnosis.

As yet the only effective treatment for this disease consists in providing bed rest and adequate supportive therapy. Surgery is of value in certain forms of the disease. Fortunately about 60 per cent of individuals infected by *C. immitis* do not develop symptoms. The remaining 40 per cent of infected humans develop symptoms that range from mild to severe, but fatalities are rare.

The motion picture illustrates the type of terrain where the fungus exists in nature and how it is isolated from soil and identified.

The route of infection and the varied symptomatology of coccidioidomycosis are described with the aid of patients and X rays. Tissue changes which occur during the course of the disease are depicted with appropriate materials. A section on the laboratory diagnosis of coccidioidomycosis illustrates isolation procedures, the gross cultural and microscopic features of *C. immitis,* and its appearance in clinical materials. The use and value of serologic tests for coccidioidomycosis are stressed.

The film was designed specifically to be of value to practicing physicians, medical students, laboratory technicians, and public health workers.

It is hoped that this brief presentation will have brought attention to teaching aids that may be of value to those concerned with furthering the training of public health workers in the field of medical mycology.

SECTION OF PHYSICS AND CHEMISTRY

SIMULATION OF GALACTIC EVOLUTION IN THE LABORATORY*

By Winston H. Bostick

Stevens Institute of Technology, Hoboken, N. J.

A technique has been developed[1,2,3] whereby ionized gas (plasma) can be projected, by magnetic forces, at speeds of 3×10^7 cm./sec. through a magnetic-field-free vacuum. The plasma has also been projected across magnetic fields in vacuum at speeds of 10^7 cm./sec. It is also possible to have a background electrically conducting medium in the form of low-pressure (about 1μ) ionized gas present in the magnetic field. This gas is photo-ionized by the ultraviolet light from the plasma gun. With the background conducting medium we can calculate, according to Spitzer,[4] the resistivity η across a strong magnetic field to be $\eta = 1.29 \times 10^{13}\ (Z\ 1m\ \Lambda)\ T^{-3/2}\ emu = 10^6\ emu$ for $T = 10^5\,^\circ K$ (electron temperature), $Z = 1$, $1m\ \Lambda = 2.3$. The magnetic Reynolds'[5] number R_m for $L = 10$ cm., and $V = 10^7$ cm./sec. is thus $Rm = 4\ \pi\ L\ V/\eta = 10^3$. Consequently, there is no doubt that with this technique we are producing magnetohydrodynamic phenomena in the laboratory.

It is appropriate to inquire concerning the fidelity with which our laboratory experiments adhere to the similarity transformations outlined by Alfven.[6] The length transformation ratio is 10^{22} in passing from the diameter of the galaxy (10^{23} cm.) to the diameter (10 cm.) of our laboratory apparatus. The transformation ratios, both desired and actual, for the other quantities involved are given in TABLE 1. It can thus be seen that, in the laboratory experiments, the time T and velocity V transform correctly. Although the electrical conductivity σ has not been transformed according to the rules it should be possible, nevertheless, to simulate cosmical magnetohydrodynamics as long as the laboratory magnetic Reynolds' number R_m is large. Although the ion density n also does not transform according to the rules, I believe that no serious defect in the simulation of the phenomena should be incurred here. It is necessary, I believe, only to have the ion density sufficiently high to make the phenomena visible by the recombination light of the ions and electrons. Gravitational effects in the galaxy will have to be simulated by inertial forces in the laboratory. There is a serious question concerning the way in which the magnetic field transforms, since its transformation ratio is out of joint by a factor of 1.6×10^{12}. However, this situation is not as hopeless as we might judge it to be at first sight, because the drift

*This paper, illustrated with lantern slides, was presented at a meeting of the Section on May 7, 1957. The experimental work described in this paper was performed at the University of California Radiation Laboratory at Livermore, Calif., and was sponsored by the United States Atomic Energy Commission, Washington, D. C.

velocities of the individual ions and electrons in the plasma across the magnetic field are given by

$$\mathbb{V}_D = \frac{c\,\mathbb{H}}{eH^2} \times \left[e\,\mathbb{E} + m\frac{d\,\mathbb{V}_D}{dt} + \mu\,\nabla H + (1/n)\,\nabla nkT \right]$$

Although there is a net value of H to the first power in the denominator, the drift velocity $\mathbb{V}_D$ of the plasma as a whole scales properly. Therefore, the various force terms in the numerator must scale with approximately the same incorrect factor as H does, in order that the drift velocities remain the same in the laboratory as in the galaxy. However, the Larmor radius will be much larger in proportion to the size of the phenomenon in the laboratory experiments than in the galaxy. However, this lack of appropriate proportion of Larmor radius to size of phenomena should not upset the simulation procedure as long as the Larmor radius of the electrons in the laboratory is small compared with the configuration being simulated. This condition is fulfilled in our laboratory

TABLE 1

SIMILARITY TRANSFORMATIONS BETWEEN THE GALAXY AND THE LABORATORY

	Length, L	Time, T	*Ideal transformation ratio*
Galaxy	10^{23} cm.	3×10^{17} sec.	
Laboratory	10 cm.	3×10^{-5} sec.	$(\text{ratio})^1 = 10^{22}$
Actual ratio	10^{22}	10^{22}	

	Particle energy	*Velocity*	*Potential*	*Current*	*Resistance*	
Galaxy		10^7 cm./sec.				
Laboratory		10^6-10^7 cm./sec.				$(\text{ratio})^0 = 1$
Actual ratio		1				

	Electric field, E	*Magnetic field, H*	*Conductivity*	*Ion Density*	
Galaxy		10^{-6} gauss		1	
Laboratory		6×10^3		10^{16}	$(\text{ratio})^{-1} = 10^{-22}$
Actual ratio		1.6×10^{-10}		10^{-16}	

	Current density	*Space charge density*	
Galaxy			
Laboratory			$(\text{ratio})^{-2} = 10^{-44}$
Actual ratio			

experiments. Furthermore, in our laboratory experiments, as in the galaxy, the gyrofrequency $eH/_{mc}$ for both positive ions and electrons exceeds the collision frequency. We thus believe that it is justifiable to simulate certain gross features of galactic formation by the transformations indicated in TABLE 1. In the interest of historical accuracy we are obliged to point out that the interesting laboratory effects were discovered first, and that the justification for using these discovered effects as simulations of galactic behavior is now attempted.

The intrinsic power and beauty of the simulation technique we are describing lies in the fact that the plasma can be photographed in three dimensions by its own recombination light. Thus, we can observe effects that are much more intricate than could be observed in plasmas even with probes or microwave techniques. It is difficult to see how the effects we observe could ever be detected in liquid sodium or mercury.

When the plasma guns are fired across a magnetic field in a good vacuum (10^{-6}–10^{-5} min. of Hg) they leave a luminous[1,3] two-line "signature" or track that can be photographed. Indeed, when two plasma guns are fired at one another the photographed tracks (FIGURE 1) show that the two pieces of plasma bounce off one another, as billiard balls do, in the center of mass system. Since the pieces of plasma retain their identity in this somewhat violent encounter they are considered to be plasma-magnetic entities, and they are accordingly called plasmoids. A diagram of the shape of the plasmoids and the currents and magnetic fields generated by the encounter is given in FIGURE 2. There is some reason[1] to believe from probe measurements that the plasmoid is a hollow cylin-

der, and it is hypothesized that the hollow structure is produced by a spin of the plasmoid.

If a plasmoid such as the one diagramed in FIGURE 2 is projected across a conducting medium (an ionized gas at 10^{-3} mm. of Hg), electromagnetic breaking action should trap the lines of magnetic field in the plasmoid. Accordingly, the DC magnetic-field lines will be dragged and twisted, and the plasmoid shape will be distorted. An attempt to suggest what might be happening is diagramed in FIGURE 3. If two such distorted plasmoids are fired at one another, as shown in FIGURE 4, it can be seen that the juxtaposition of the apexes of the plasmoids could bring about stagnation points in the magnetic field, and the magnetic field lines could hop from one plasmoid to the other, thus splicing the two plasmoids together. This picture of FIGURE 4 is an attempt to explain the peculiar experimental effects shown in FIGURES 5, 6, 7, and 8 where distorted plasmoids can be seen to glue on to one another, head-to-head, but studiously repel one another on a flank-to-flank encounter.

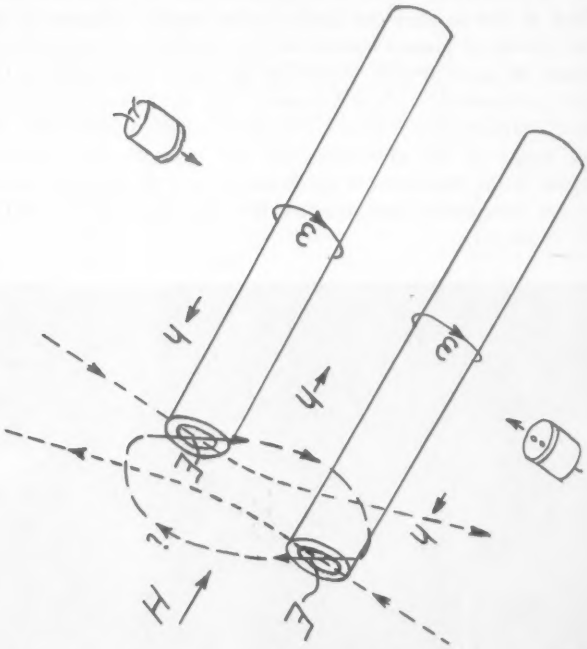

FIGURE 2. Diagram of two plasmoids encountering each other as they travel across a magnetic field in a good vacuum, as in FIGURE 1. There is evidence that the plasmoid is a cylinder that elongates in the direction of the magnetic field as it proceeds across the magnetic field. Evidence also suggests that the cylinder is hollow and spins. The spin is indicated in the diagram by ω.

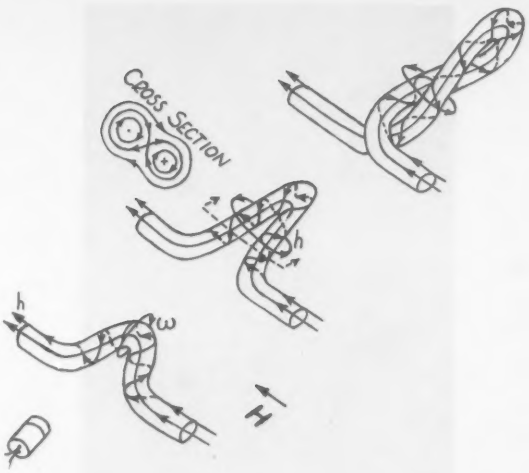

FIGURE 3. Suggested configuration of plasma and magnetic field of a single plasmoid projected across a magnetic field when an electromagnetic braking action occurs because of a pressure of about 1μ in the vacuum chamber. The twisted configuration will itself assume a general helical configuration (try twisting two strands of wire, rope, or rubber tightly). This helical configuration is seen especially clearly in FIGURE 5.

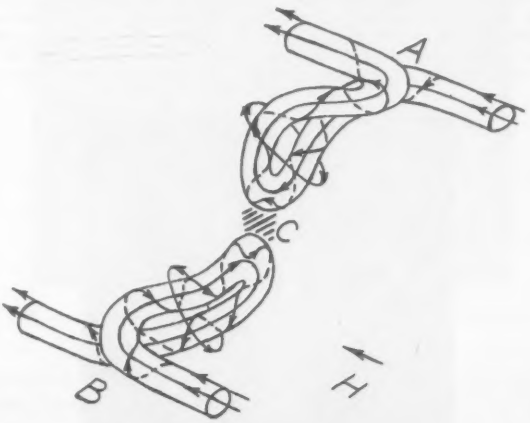

FIGURE 4. Suggested configurations of plasma and magnetic fields existing in the formation of a barred spiral. The stagnation point produced when the two leading loops of the plasmoids approach one another (region C) permits the lines of force to leap from one plasmoid to another, carrying plasma across and tying the two plasmoids together.

FIGURE 5. A sequential study of barred spirals produced by firing 2 plasmoids from sources 10 cm. apart at one another simultaneously across a magnetic field of 4800 gauss. The Kerr-cell exposure times are 2 μsec., and the various delay times of the sequence are indicated in μsec. The pressure in the chamber is 2 μ. The plasmoids and their trajectories are rendered luminous primarily by the recombination light of the titanium and deuterium ions that come from the plasma source. The photographs on the left are the left stereoscopic photographs; those on the right, the right stereoscopic photographs, with an angle between the two views of 10°. The middle photograph is taken straight ahead along the direction of the magnetic field. The current (3000 amperes for 0.4 sec.) through the source produces a magnetic field that opposes the DC field and diminishes the velocity of projection of the plasmoid across the magnetic field.

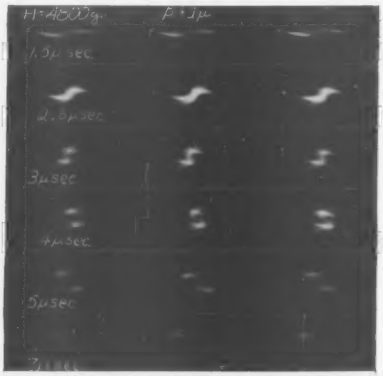

FIGURE 6. The same as FIGURE 5, except that the pressure in the chamber is 1 μ.

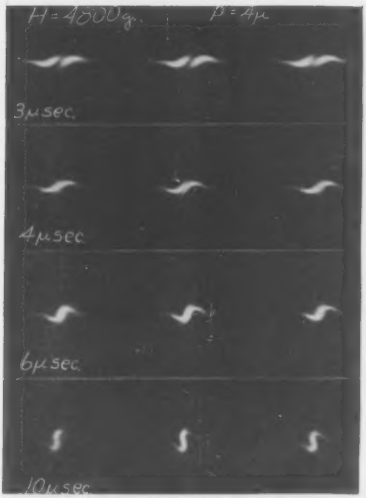

FIGURE 7. The same as FIGURE 5, except that the pressure is 4 μ.

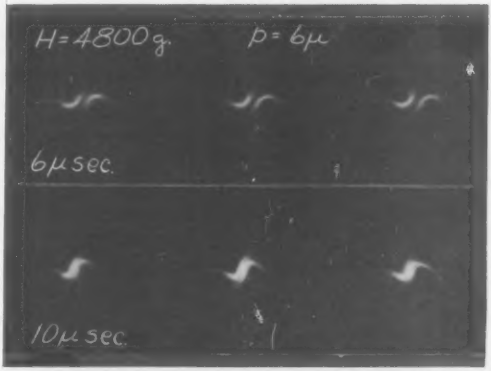

FIGURE 8. The same as FIGURE 5, except that the pressure is 6 μ.

It is possible to fire two plasmoids at one another and to have them fasten head-to-tail to form a flattened ring that flips into a figure 8, as shown in FIGURE 9.

It is also possible to fire four or eight plasmoids at one another and produce the spirals that end up as rings, as shown in FIGURES 10, 11, and 12. In FIGURES 10 and 11 the ring remains stationary in the magnetic field. In FIGURE 12 the ring moves toward the camera. An attempt

FIGURE 9. Sequence of photographs of formation of a flattened ring which flips into a figure 8. The ring is formed by firing 2 sources across a magnetic field (into the paper) of 2800 gauss with a pressure of 2.2 μ in the chamber. The exposure time is 2 μsec., and the various delay times are indicated in μsec. The current in the source is in such a direction as to diminish the velocity of propagation in the magnetic field.

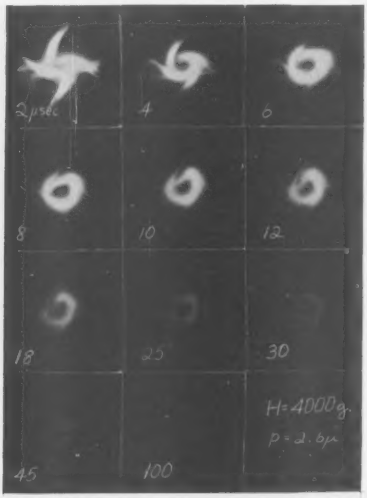

FIGURE 10. An example of the formation of a ring by firing 4 sources across a magnetic field of 4000 gauss (into the paper) at a pressure of 2.6 μ. The exposure time is 2 μsec., and the various delay times are indicated in μsec. The current in the sources is in such a direction that the velocity of projection of the plasma is retarded by the DC magnetic field. The 100-μsec. delay photograph actually shows a faint ring on the original.

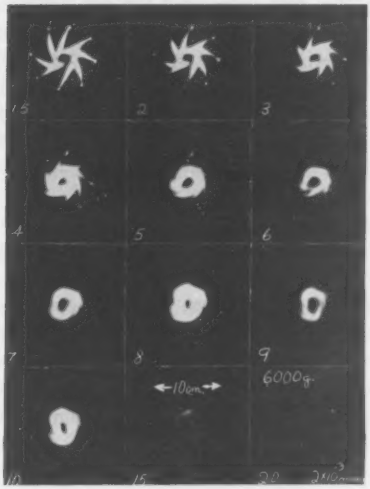

FIGURE 11. Photographic sequence of the formation of a ring from the plasmoids from 8 sources fired across a magnetic field of 6000 gauss into the paper. The pressure is 2 μ. The exposure time is 2 μsec., and the delay times are indicated in μsec. The current in the sources is in the direction to diminish the velocity of projection of the plasmoids across the DC magnetic field. Note that at 15-μsec. delay, the ring has grown an ear.

to describe what is happening in FIGURE 12 is diagramed in FIGURE 13. We hypothesize that the rings of FIGURES 10 and 11 are actually two rings, as in FIGURE 13, but that they are so topologically intertwined that they cannot separate. In fact, we would go so far as to suggest that the ring configurations of FIGURES 12 and 13 are actually an approximation to a force-free configuration of magnetic fields and currents.

On the basis of what plasmoids in the laboratory have taught us, we can now construct in outline a story of the origin of the galaxy in which the spiral arms are created. Although the story at this stage of the development of the subject will be necessarily only qualitative and imperfect, I nevertheless believe that the plasmoids have given us the basic clue to the origin of the spiral arms.

The starting point of the story is a region of space approximately 10^{25} cm. in diameter, or greater. This space is uniformly populated by equal numbers of protons and electrons, that is, ionized hydrogen plasma. Statistical fluctuations in density will produce incipient gravitational centers of attraction in regions where the density is somewhat higher than in surrounding regions. As Jeans has shown, this process is essentially the e-folding of a gravitational instability, and the matter will

end up in clumps. The new ingredient we are adding to Jeans' picture is the magnetic field that might be generated by this process. It is quite possible that alignment of electron spins and cooperative interaction of the electric currents carried by both electrons and positive ions may lead to an incipient magnetic field. It is difficult to show how this magnetic field, in the beginning, could grow in crescendo fashion by regenerative dynamo action, although we can make out a fair case for the existence of dynamo action during a later phase of the galaxy.

We shall assume that the magnetic field of this region of higher density is able to *inhibit*, to a certain extent, motion of plasma *across* the magnetic field. However, the plasma will be free to travel *along* the lines of magnetic force as if they were not there. Hence, the plasma by self-gravitational contraction can be expected to form the pancake or lenticular shaped concentration which is characteristic of a galaxy looked at edge-on (FIGURE 14a). We now have a perfect setup for Rayleigh-Taylor instability to occur, with the plasma being supported against this central gravitational field by the magnetic field. The plasma may be ex-

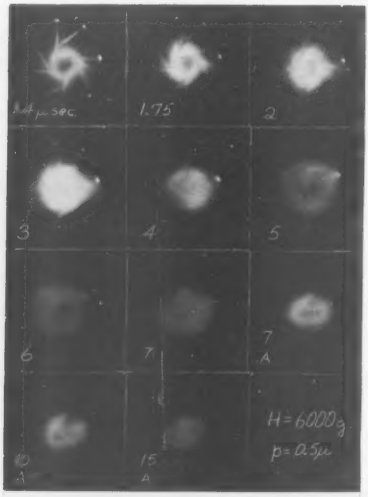

FIGURE 12. Sequence of photographs taken in a manner similar to those of FIGURE 11 except that the source current is in the direction which aids the velocity of propagation of the plasmoid across the magnetic field, and the pressure is 0.5 μ. The whirl ring that is formed gets progressively out of focus as it apparently moves toward the camera and, at 7-μsec. delay, it is badly blurred. The delay times "7A, 10A, and 15A" correspond to moving the camera back 15 cm. which, at least for 7A, sharpens up the picture. It is believed that for delays 10A and 15A, and perhaps for 7A, the ring has traveled until it has encountered the Lucite window of the vacuum system, which is 20 cm. from the position at which the ring is formed.

pected to jet inward (FIGURE 14b) carrying, distorting, and twisting the lines of force even as our plasmoids apparently do in the laboratory in FIGURES 5, 6, 7, 8, 10, 11, and 12 and as hypothesized in FIGURES 3, 4, and 13. Indeed, the laboratory experiments in which we project plasma across a magnetic field are essentially a demonstration of this form of Rayleigh-Taylor instability: the magnetic field attempts to slow down the plasma that proceeds from the gun. As the plasma is initially decelerated by the magnetic field and by any subsequent electromagnetic breaking forces, it feels a fictitious gravitational force in the direction in which it was projected; the plasma forms a jet that moves across the magnetic field as shown, for example, in FIGURES 5, 6, 7, and 8.

We should expect these instability jets in the galaxy to spiral in toward the center (FIGURE 14b) as they do in the laboratory experiments. Presumably, if there are only two jets, they will form a barred spiral galaxy (as in our laboratory case, for example, of FIGURES 5 and 6), but several jets will form a several-armed spiral (FIGURES 9, 10, 11, and 12). Thus, we draw the sequence of FIGURES 14a, b, c, and d, which indicates our picture of galaxy formation. The new population type I stars are presumably condensed out of the hydrogen plasma that has been concentrated in the arms. These stars, which derive their nuclear energy from the H-H cycle, are those that form the major portion of the population of these arms and thereby lend the arms their luminous structure. It can be seen from the diagrams of FIGURES 3, 4, and 14 that the magnetic field of such a Taylor-instability jet that has become a spiral arm will be predominantly parallel to the arm. Chandrasekhar and Fermi[7,8] have shown that a magnetic field of 10^{-6} gauss in the arms is necessary to prevent the complete gravitational collapse of the ionized hydrogen present in the arms.

We can now make plausible a magnetic-field and electric-current pattern that can possibly lead to regenerative dynamo action in the galaxy, and hence to a galactic magnetic field that grows in time as gravitational energy is transformed into magnetic energy, as shown in FIGURE 14d. The electromotive force or electric field that drives the currents from the periphery of the arms along the arms toward the center in the galaxy (FIGURE 14d) is strictly analogous to the electromotive force that must exist in the laboratory barred spiral (FIGURE 5) where the configuration can be observed to rotate in a magnetic field. The current configuration diagramed in FIGURE 14d suggests a manner in which the dipole field of the galaxy can be reinforced by the currents that are driven along the arms and that return from the center to the periphery by flowing through the galactic halo.

If the general dipole field of the galaxy continues to rise in magnitude, any electrically conducting material (hydrogen plasma) in its neighborhood will have induced within it currents whose magnetic field will be

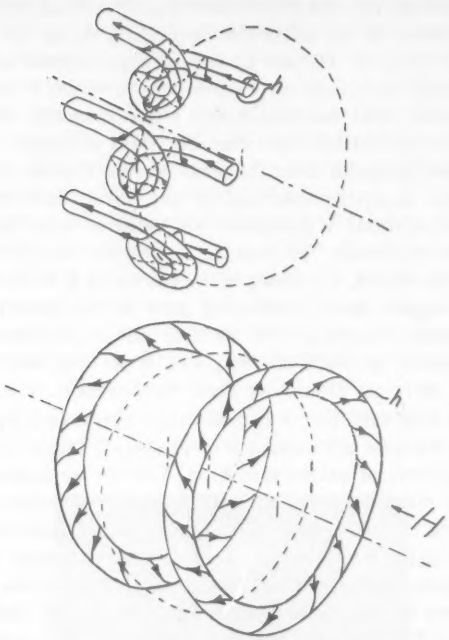

FIGURE 13. Suggested description of formation of two whirl rings that move away from one another. It is believed that one of these rings has been observed in FIGURE 12.

repelled by the galaxy. If this hydrogen plasma represents an incipient galaxy in proximity to the other galaxy, it will be repelled by the latter and vice versa.

Indeed, we can make a rough estimate of this velocity of recession that will be produced by this magnetic repulsion between galaxies. If the magnetic dipole field strength within the galaxy is about 5×10^{-6} gauss, and if the effective volume occupied by this field (for purposes of energy calculation) is about 25 times that of the galactic disk, the magnetic energy available per cm.^{3} of the galactic disk for acceleration of the galaxy will be $25 \times (5 \times 10^{-6})^2 \ (1/8\pi)$ ergs $\cong 25 \times 10^{-12}$ ergs. If the acceleration of one galaxy with respect to the other involves a mass corresponding to one hydrogen ion pair per cm.^{3} in each galaxy, and if this magnetic energy for each galaxy is transformed to kinetic energy with respect to the center of mass of the two galaxies, the velocity of recession of each galaxy from the common center of mass is 5.6×10^6 cm./sec. The velocity of recession of one galaxy from the other is twice this speed, or $11 \times$

10^6 cm./sec. = 110 km./sec. It is to be emphasized that this estimate of speed probably represents a lower limit on the speed to be expected because, when the two galaxies are young and in close proximity, it is quite possible that the magnetic fields in the region between the centers of the two galaxies are compressed to values higher than 5×10^{-6} gauss. Hence, there is a very real possibility that the kinetic energies of the galaxies with respect to one another are produced by magnetic repulsion. It is presumably this kinetic energy that produces the expansion of the universe, the galaxies on the periphery receding more rapidly than those that are close. It is interesting to note that the high plasma speeds that we have been able to generate in the laboratory with the plasma gun have been produced essentially by the process of magnetic repulsion.

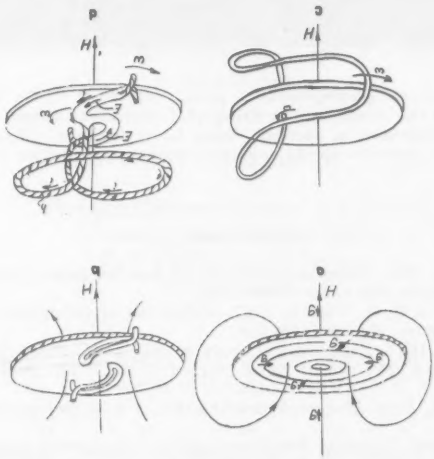

FIGURE 14. Hypothesis on the process of formation of a barred spiral galaxy: (a) the pancake or lenticular shape produced by the gravitational attraction of a hydrogen plasma (protons and electrons) in conjunction with the magnetic field shown; (b) the origin of the arms produced by plasma jets moving across the magnetic field, the jets being presumably caused by Taylor instability; (c) a suggestion as to how a tube of magnetic flux of a dipole field will be dragged by the differential rotation in the galaxy; and (d) a suggestion as to how regenerative dynamo action can occur in the galaxy to produce a galactic magnetic field which increases as time goes on (electric current flow from galactic center to periphery via the galactic halo is indicated to show how the initial magnetic field H can be reinforced).

In FIGURE 15 we reproduce several examples of barred spirals photographed in the laboratory. Not only do these photographs bear striking two-dimensional resemblances to some of the astronomical barred spirals but, in addition, they exhibit (as do FIGURES 5, 6, 7, and 8) the characteristic that the arms are corkscrew shaped. In a given laboratory barred

spiral, both arms will be either right-handed or left-handed. This lack of coplanarity of the arms has been observed in astronomical spiral galaxies. Also visible in some of the laboratory barred spirals are the bifurcated tails on the arms, especially in FIGURE 6. It is worth noting that split tails are frequently observed in astronomical spiral galaxies.

FIGURE 15. Kerr-cell photographs (2 μsec. exposure) of the recombination light resulting from the simultaneous firing of 2 plasmoids across the magnetic fields indicated. Pressures in microns; delay times in μsec. The barred spiral configuration shown here is easily produced over a wide range of parameters.

References

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CONFERENCES HELD

SECTION OF BIOLOGY

ANIMAL DISEASE AND HUMAN HEALTH

Wednesday, September 11
Thursday, September 12, and
Friday, September 13, 1957

Conference Chairman: James Lieberman
Communicable Disease Center, U. S. Public Health Service,
Atlanta, Ga.

WEDNESDAY, SEPTEMBER 11, 1957

Session Chairman: Robert J. Anderson
Communicable Disease Center
U. S. Public Health Service
Atlanta, Ga.

9:00 A.M. –
Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; New York University, New York, N. Y.

"Animal Diseases – Their Relationship to the Health of Man" – J. D. Martin, Louisiana State Health Department, New Orleans, La.

"Orphan Viruses of Man and Animal" – G. D. Hsiung and Joseph L. Melnick, Yale University School of Medicine, New Haven, Conn.

"Possible Significance of Human Antiviral Neutralizing Substances in Animal Sera" – Morton Klein, Temple University, Philadelphia, Pa.

"Counterparts of Human Viral Disease in Animals" – Hilary Koprowski, Wistar Institute, University of Pennsylvania, Philadelphia, Pa.

Open Discussion

12:30 P.M. –
Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.
"Animal Disease and Biological Warfare Defense" – Lt. Col. Jack H. Hempy, Office of the Surgeon General, USAF, Washington, D. C.

Session Chairman: Morris Schaeffer
Communicable Disease Center
U. S. Public Health Service
Montgomery, Ala.

2:00 P.M. –
Film – "An Introduction to Arthropod-Borne Encephalitis"

Symposium: "The Viral Encephalitides" – William McD. Hammon, University of Pittsburgh, Pittsburgh, Pa., *Moderator.*

"Natural History of Viral Encephalomyelitis in the United States" –

"Western" – Archie D. Hess, Communicable Disease Center, U. S. Public Health Service, Logan, Utah.

"Eastern" – Preston Holden, Communicable Disease Center, U. S. Public Health Service, Greeley, Colo.

"Vector Relationships in the Arthropod-Borne Encephalitides" – Roy W. Chamberlain, Communicable Disease Center, U. S. Public Health Service, Montgomery, Ala.

"Host Relationships of the Arthropod-Borne Encephalitides" – Robert E. Kissling, Communicable Disease Center, U. S. Public Health Service, Montgomery, Ala.

"Use of Equine Encephalomyelitis Vaccine in the Prevention of Encephalitis in Pheasants Under Epidemic and Pre-Epidemic States" – Oscar Sussman, Daniel Cohen, and Jean H. Gerende, New Jersey State Health Department, Trenton, N. J.

"The Changing Picture of Murine Typhus in the United States" – Harry D. Pratt, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

"The Epidemiology of Q Fever" – Hartwell H. Welsh, California State Health Department, Berkeley, Calif., and Martin M. Kaplan, World Health Organization, Geneva, Switzerland.

Open Discussion

5:30 P.M. –
Cocktail Hour – Academy Building

6:30 P.M. –
Dinner – Academy Building
"Global Priorities in Animal Disease Control" – Martin M. Kaplan, World Health Organization, Geneva, Switzerland.

"The Pan American Zoonoses Center" – Benjamin D. Blood, Pan American Sanitary Bureau, Buenos Aires, Argentina.

THURSDAY, SEPTEMBER 12, 1957

Session Chairman: Martin M. Kaplan
World Health Organization, Geneva, Switzerland

9:00 A.M. –

Symposium: "Leptospirosis – A Contemporary Problem" – Donald S. Martin, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga., *Moderator.*

"Animal Reservoirs" – B. Babudieri, Instituto Superióre di Sanità, Rome, Italy.

"Infections in Man" – Theodore L. Woodward, University of Maryland, Baltimore, Md.

"Laboratory Diagnosis" – William S. Gochenour, Chester A. Gleiser, and Martha K. Ward (U. S. Public Health Service), Walter Reed Army Medical Center, Fort Detrick, Md.

"Epidemiological Patterns" – Mildred M. Galton, Robert W. Menges, and James H. Steele, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

Symposium: "Ornithosis (Psittacosis) in Domestic Fowl" – Justin M. Andrews, National Institutes of Health, Public Health Service, Bethesda, Md., *Moderator.*

"Newer Findings in Turkeys" – John P. Delaplane, Agricultural and Mechanical College of Texas, College Station, Texas.

"Newer Methods for Detection of Avian Ornithosis" – Albert A. Benedict, University of Kansas, Lawrence, Kan.

"Preventive Chemotherapy – Advantages and Limitations" – Karl F. Meyer, The George Williams Hooper Foundation, San Francisco, Calif.

Open Discussion

12:30 P.M. –

Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.

Session Chairman: Martin P. Hines
North Carolina State Board of Health
Raleigh, N. C.

2:00 P.M. –

Symposium: "Recent Developments in the Epidemiology of Rabies" –

Ernest S. Tierkel, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga., *Moderator.*

"Bat Rabies – Experimental Host Transmission Studies" – Kenneth F. Burns, Fourth U. S. Army Medical Laboratory, Fort Sam Houston, Texas.

"Newer Tools for the Prevention of Rabies in Domestic Animals" – M. L. Kaeberle, University of Illinois, Champaign, Ill.

"Prophylaxis Against Rabies in Humans – 1957" – John P. Fox, Tulane University, New Orleans, La.

"Brucellosis – Present and Future" – Norman B. McCullough, National Institutes of Health, Public Health Service, Bethesda, Md., and C. K. Mingle, U. S. Department of Agriculture, Washington, D.C.

Open Discussion

5:30 P.M. –
Cocktail Hour – Academy Building

FRIDAY, SEPTEMBER 13, 1957

Session Chairman: Earl C. Chamberlayne
Pan American Sanitary Bureau
Washington, D.C.

9:00 A.M. –
"Animal Parasites and Human Disease" – Thomas W. M. Cameron, McGill University, Montreal, Canada.

"Industrial Anthrax" – Philip S. Brachman and F. Robert Fekety, Communicable Disease Center Activities, U. S. Public Health Service, University of Pennsylvania, Philadelphia, Pa.

"Newcastle Disease" – R. P. Hanson, University of Wisconsin, Madison, Wis., and C. A. Brandly, University of Illinois, Urbana, Ill.

"Controlling *Salmonella* Infections of Animal Origin" – Philip R. Edwards, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

"The Exotic Zoonoses" – Maurice S. Shahan and Jacob Traum, Plum Island Animal Disease Laboratory, U. S. Department of Agriculture, Greenport, N. Y.

"Listeriosis – A Potential Public Health Problem" – John F. Winn, W. B. Cherry and Elizabeth O. King, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

"Public Health Importance of Animal Tuberculosis" – Robert J. Anderson, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

12:30 P.M.–
Luncheon – Academy Building, 2 East 63rd Street, New York, N.Y.

Session Chairman: Ralph S. Muckenfuss
Naval Medical Research Institute
Bethesda, Md.

2:00 P.M. –
"Recent Developments in Animal Ringworm and Their Public Health Implications" – William Kaplan, Lucile Georg, and Libero Ajello, Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga.

"Comparative Studies on Viral Hepatitis in Animals and Man" – Morris Pollard, University of Texas Medical Branch, Galveston, Texas.

"Cat Scratch Fever" – James E. Prier, Merck Sharp & Dohme, West Point, Pa.

"New Knowledge on the Ecology of Sylvatic Plague" – Leo Kartman, Frank M. Prince, Stuart F. Quan, and Harold E. Stark, Communicable Disease Center, U. S. Public Health Service, San Francisco, Calif.

"A Comparison of Rheumatoid-Like Arthritis in Swine and Rheumatoid Arthritis in Man" – Dennis Sikes, University of Georgia, Athens, Ga.

"Pulmonary Adenomatosis in Sheep and Possible Relationship to Cancer in Humans" – Francisco Duran-Reynals and Keen A. Rafferty, Yale University School of Medicine, New Haven, Conn.

SUMMARY OF CONFERENCE

Leonard M. Schuman
University of Minnesota
Minneapolis, Minn.

Open Discussion

ORGANIZING COMMITTEE

James Lieberman, *Chairman*

Robert L. Burkhart
Thomas W. M. Cameron
David L. Coffin
William H. Feldman
William A. Hagan
Morris Pollard
James H. Steele

NEW STEROID COMPOUNDS WITH PROGESTATIONAL ACTIVITY

Monday, October 7 and
Tuesday, October 8, 1957

Conference Co-Chairmen:

Abraham E. Rakoff, Jefferson Medical College
Philadelphia, Pa.

Edward C. Reifenstein, Jr., The Squibb Institute
for Medical Research, New York, N. Y.

MONDAY, OCTOBER 7, 1957

Session Chairman: Abraham E. Rakoff
Jefferson Medical College, Philadelphia, Pa.

9:00 A.M. –

Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; New York University, New York, N. Y.

Introduction to the Conference – Abraham E. Rakoff, Jefferson Medical College, Philadelphia, Pa.

BIOLOGIC ACTIVITY IN ANIMALS

"Some Biological Activities in Animals of Certain Progestagens" –

I. "17-α-Hydroxyprogesterone-17-n-Caproate" – W. B. Kessler and A. Borman, The Squibb Institute for Medical Research, New Brunswick, N. J.

II. "9- and 12-Halo-11-Oxygenated Progesterones" – J. Fried, W. B. Kessler, and A. Borman, The Squibb Institute for Medical Research, New Brunswick, N. J.

"Some Chemical and Biological Properties of 19-Nor-17α-Ethynyltestosterone" – Daniel A. McGinty, Parke, Davis & Company, Detroit, Mich.; and Carl Djerassi, Syntex, S. A., Mexico, D.F., Mexico.

"Some Biological Activities of 17-Ethynyl and 17-Alkyl Derivatives of 17-Hydroxyestrenones" – Victor A. Drill and Francis J. Saunders, G. D. Searle & Company, Chicago, Ill.

"The Comparative Potency of Newer Progestogenic Compounds in Animals as Indicated by Different Biologic Tests" – M. X. Zarrow, L. E. Peters and A. L. Caldwell, Jr., Purdue University, Lafayette, Ind.

PREGNANCY IN ANIMALS

"The Biologic Actions of 17-α-Hydroxyprogesterone-17-n-Caproate on the Reproductive Processes of the Rat" – J. T. Velardo, Yale University School of Medicine, New Haven, Conn.

"Attempts to Induce and Maintain Pregnancy in Prepuberal Mice by Means of 17-α-Hydroxyprogesterone-17-n-Caproate" – M. Smithberg, College of Medicine, University of Florida, Gainesville, Fla.

12:30 P.M. –
Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.

Session Chairman: Edward C. Reifenstein, Jr.
The Squibb Institute for Medical Research, New York, N. Y.

2:00 P.M. –

BIOLOGIC ACTIVITY IN MAN

"The Comparative Pharmacologic and Clinical Activity of 19-Nortestosterone and 17-Hydroxyprogesterone Derivatives in Man" – Jeanne A. Epstein and Herbert S. Kupperman, New York University-Bellevue Medical Center and Beth Israel Hospital, New York, N. Y.

METABOLIC ACTIVITY

"The Effect of 17-α-Hydroxyprogesterone-17-n-Caproate on Reproductive Performance and Growth Fattening of Cattle" – F. X. Gassner, Colorado State University, Fort Collins, Colo.; K. R. Johnson, University of Idaho, Moscow, Idaho; A. V. Nalbandov, University of Illinois, Urbana, Ill.; and E. C. Reifenstein, Jr., The Squibb Institute for Medical Research, New York, N. Y.

"The Metabolic Effects in Man of Steroids with Progestational Activity" – Richard L. Landau, Kathleen Lugibihl and Dean F. Dimick, Department of Medicine, University of Chicago, Chicago, Ill.

ENDOMETRIAL RESPONSE IN MAN

"The Comparative Activity of Progestational Agents on the Human Endometrium and Vaginal Epithelium of Surgical Castrates" – G. L. Wied and M. E. Davis, University of Chicago, Chicago, Ill.

"Histochemical Reactions in the Human Endometrium Following Administration of 17-Ethynyl-Estreneolone" – P. N. Shah, M. E. Long and

A. L. Southam, Columbia University College of Physicians and Surgeons, New York, N. Y.

"The Effects of Various Progesterone Compounds on the Normal Preovulatory Endometrium as Compared with the Endometrium in Secondary Amenorrhea" – M. James Whitelaw, University of California School of Medicine, San Francisco, Calif.

"The Effects of 17-Ethynyl-Estreneolone on the Human Endometrium" – M. Roland, Albert Einstein College of Medicine, New York, N. Y.

"The Effects of Progestational Compounds on the Reproductive Processes of the Human Male" – Carl G. Heller and W. M. Laidlaw, University of Oregon Medical School, Portland, Ore.; and Warren O. Nelson, The Rockefeller Institute for Medical Research, New York, N. Y.

5:30 P.M. –
Cocktail Hour – Academy Building

7:00 P.M. –
Dinner by Invitation – Academy Building

TUESDAY, OCTOBER 8, 1957

Session Chairman: Warren O. Nelson
The Rockefeller Institute for Medical Research, New York, N. Y.

9:00 A.M. –

GYNECOLOGIC PROBLEMS

"A Comparative Study of the Effect of the Progestational Agents in Human Menstrual Abnormalities" – A. L. Southam, Columbia University College of Physicians and Surgeons, New York, N. Y.

"The Effects of Certain 19-Nor Steroids upon the Reproductive Processes of Man" – G. Pincus, Worcester Foundation for Experimental Biology, Shrewsbury, Mass.; and J. Rock and C. R. Garcia, Fertility and Endocrine Clinic and the Reproductive Study Center of the Free Hospital for Women in Brookline, Mass.

"The Clinical Application of the New Long-Acting Progestational Steroid, 17-α-Hydroxyprogesterone-17-n-Caproate" – J. J. Gold, Michael Reese Hospital and Chicago Medical School, Chicago, Ill.; and M. k. Cohen, Michael Reese Hospital, Chicago, Ill.

INFERTILITY

"The Clinical Use of New Progestational Steroids in Infertility" – E. T. Tyler and H. J. Olson, University of California at Los Angeles School of Medicine, Los Angeles, Calif.

"The Effects of New Progestational Agents in Human Gynecologic and Adrenal Disorders" – S. L. Clark and R. B. Greenblatt, Medical College of Georgia, Augusta, Ga.

"Evaluation of the Biologic Activities in Man of 17-α-Hydroxyprogesterone-Acetate, an Oral Progestational Compound" – Joseph W. Goldzieher, Department of Endocrinology, Southwest Foundation for Research and Education, San Antonio, Texas; William F. Peterson, Washington, D. C.; and Richard A. Gilbert, Chevy Chase, Md.

12:30 P.M. –
Luncheon – Academy Building, 2 East 63rd Street, New York, N.Y.

Session Chairman: C. Lee Buxton
Yale University School of Medicine, New Haven, Conn.

2:00 P.M. –
"Observations on the Role of Progestational Agents in Human Gynecologic Disorders and Pregnancy Complications" – Hanns-Werner Boschann, Department of Obstetrics and Gynecology, Moabit Hospital, Free University of Berlin, Berlin, West Germanv.

OBSTETRICS: ABORTION

"High Potency Progestational Agents in Human Pregnancy" – C. Paul Hodgkinson, Eli J. Igna, and Alfred P. Bukeavich, Henry Ford Hospital, Detroit, Mich.

"Clinical Observations on the Use of 17-Ethynyl-19-Nortestosterone in Repeated and Threatened Abortions" – Daniel Abramson, Boston Lying-In Hospital, Department of Obstetrics, Harvard Medical School, Boston, Mass.

"The Clinical Use of 17-α-Hydroxyprogesterone-17-n-Caproate in Habitual Abortion" – E. C. Reifenstein, Jr., The Squibb Institute for Medical Research, New York, N.Y.

OBSTETRICS: OTHER PROBLEMS

"The Clinical Use of 17-α-Hydroxyprogesterone-17-n-Caproate in the Prevention of Premature Labor, and Its Effect upon Fetal Salvage" – Eduard Eichner, Mount Sinai Hospital, Cleveland, Ohio.

"The Treatment of Human Diabetic Pregnancies with 17-α-Hydroxyprogesterone-17-n-Caproate" – L. Gillespie, L. I. Sexton and P. White, New England Deaconess Hospital, Boston, Mass.

SUMMARY OF CONFERENCE

Abraham E. Rakoff
Jefferson Medical College, Philadelphia, Pa.

ORGANIZING COMMITTEE

Abraham E. Rakoff
Edward C. Reifenstein, Jr.
C. Lee Buxton
J. William Crosson
John E. Gajewski
James H. Leathem
Warren O. Nelson

GENETIC CONCEPT FOR THE ORIGIN OF CANCER

Friday, October 25 and
Saturday, October 26, 1957

Conference Chairman: Leonell C. Strong
Biological Station of Roswell Park Memorial Institute
Springville, N. Y.

FRIDAY, OCTOBER 25, 1957

Session Chairman: Clarence C. Little
Jackson Memorial Laboratory, Bar Harbor, Me.

9:00 A.M. –
Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; New York University, New York, N. Y.

"Genetic Concept for the Origin of Cancer: Historical Review" and "Origin of Inbred Strains of Mice" – Leonell C. Strong, Biological Station of Roswell Park Memorial Institute, Springville, N. Y.

"Effects of Selection on Chemically Induced Tumors in Mice" – Leonell C. Strong, Biological Station of Roswell Park Memorial Institute, Springville, N. Y., and L. D. Sanghvi, Indian Cancer Research Center, Bombay, India.

"Chemically Induced Carcinomas of the Mammary Gland and Fibrosarcoma with and without Selection" – Hugh F. O'Neill, Mercy Hospital, Buffalo, N. Y.

"Maternal Age Selection and Susceptibility to Chemically Induced Tumors" – Ross Markello, University of Buffalo School of Medicine, Buffalo, N. Y.

"Genetic Studies on Tumors in Mice" – Walter Heston, National Cancer Institute, Public Health Service, Bethesda, Md.

"Genetic Concepts in Mammary Cancer in Mice" – J. J. Bittner, University of Minnesota, Minneapolis, Minn.

"Genetic Studies in Experimental Neoplasms" – Lloyd Law, National Cancer Institute, Public Health Service, Bethesda, Md.

12:30 P.M. –
Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.

Session Chairman: T. S. Hauschka
Roswell Park Memorial Institute, Buffalo, N. Y.

2:00 P.M. –
"Malignancy and the Genetics of the Somatic Cell" – Jack Schultz, Institute of Cancer Research, Philadelphia, Pa.

"Genetic Studies on Tumor Immunity" – D. B. Amos, Roswell Park Memorial Institute, Buffalo, N. Y.

"Chromosomal Evidence for Genetic Variation in Tumors" – Albert Levan, University of Lund, Lund, Sweden.

"Chemically Induced Imitations of Mitotic Anomalies Common to Cancer Cells" – John J. Biesele, Sloan-Kettering Institute, New York, N.Y.

"Cellular Aspects of Tumorigenesis" – Walter Burdette, St. Louis University Medical School, St. Louis, Mo.

"Carcinogenic Mutagens" – R. W. Barratt, Dartmouth College, Hanover, N. H., and E. L. Tatum, Rockefeller Institute for Medical Research, New York, N. Y.

5:30 P.M. –
Cocktail Hour – Academy Building

SATURDAY, OCTOBER 26, 1957

Session Chairman: Hilary Koprowski
Wistar Institute, Philadelphia, Pa.

9:00 A.M. –
"Mutation Changes in Viruses" – S. A. Luria, University of Illinois, Urbana, Ill.

"Hormones in Cancer and the Genetic Concept" – William U. Gardner, Yale University, New Haven, Conn.

"Relationships, Established and Prospective, Between Viruses and Cancer" – Wendell Stanley, University of California, Berkeley, Calif.

"Evidence from Experimentally Induced Neoplasms Relative to the Mutation Hypothesis of the Origin of Malignant Disease" – W. F. Dunning, University of Miami, Coral Gables, Fla.

"The Biological Action of Ionizing Radiation, the Aging Process, and Cancerogenesis" – G. Failla, College of Physicians and Surgeons, Columbia University, New York, N. Y.

"Activity of Human Cells" – Theodore T. Puck, University of Colorado Medical Center, Denver, Colo.

"Cellular Changes Induced by Radiation" – Charles M. Pomerat, University of Texas, Medical Branch, Galveston, Texas.

"Morphogenesis" – Edmund W. Sinnott, Yale University, New Haven, Conn.

12:30 P.M. –
Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.

Session Chairman: P. C. Koller
Chester Beatty Research Institute, London, England

2:00 P.M. –
"Mammalian Cytogenetics and the Cancer Problem" – Allen Griffen, Jackson Memorial Laboratory, Bar Harbor, Me.

"Genetic Plant Tumors in Nicotiana" – Harold H. Smith, Cornell University, Ithaca, N. Y.

"Genetic Consideration of Leukosis Tumor Growth in Poultry" – John W. Gowen, Iowa State College, Ames, Iowa.

"ABO Blood Groups in Man and Cancer" – L. D. Sanghvi, Indian Cancer Research Center, Bombay, India.

"Genetics of Human Cancer: A Survey of Methods" – Peter Gorer, Guys Hospital, London, England.

"Studies on Human Cancer Families" – C. P. Oliver, University of Texas, Austin, Texas.

"Induction, Organization, and Differentiation of Cells" – J. F. D. Holtfreter, University of Rochester, Rochester, N. Y.

"Genetics of Homeostasis" – T. Dobzshansky, Columbia University, New York, N. Y.

"Mutations in Genetics" – H. J. Muller, University of Indiana, Bloomington, Ind.

SUMMARY OF CONFERENCE

C. C. Little
Jackson Memorial Laboratory, Bar Harbor, Me.

THE DEVELOPMENT OF HIGH SCHOOL STUDENTS TOWARD CAREERS IN BIOLOGY

Friday, October 18 and
Saturday, October 19, 1957

Conference Co-Chairmen
George Schwartz, Forest Hills High School
Forest Hills, N.Y.
Leon Rintel, Forest Hills High School
Forest Hills, N.Y.

FRIDAY, OCTOBER 18, 1957

Session Chairman: Walter Wolff
William Cullen Bryant High School, Long Island City, N. Y.

7:30 P.M. –

Greetings from the Academy – A. S. Gordon, Chairman, Section of Biology, The New York Academy of Sciences, New York, N. Y.; New York University, New York, N. Y.

Introduction – "Opportunities for Research in Biology by High School Students" – Walter Wolff, William Cullen Bryant High School, Long Island City, N. Y.

"The Use of High School Students as Assistants in Research" – Myron Gordon, American Museum of Natural History, New York, N. Y.

"Summer Employment of Science Students in Industry and Research Laboratories" – Samuel Schenberg, Board of Education of the City of New York, N. Y.

"A Marine Museum and Laboratory for High School Teachers and Students" – Erwin Ernst, Setauket High School, Setauket, Long Island, N.Y.

"Work in Herpetology with High School Students" – James Oliver, New York Zoological Society, New York, N. Y.

"Research in Biology in Every High School" – Maurice Bleifeld, Benjamin Franklin High School, New York, N. Y.

Discussion and Summation – David Sessler, Long Island City High School, Long Island City, N. Y.

SATURDAY, OCTOBER 19, 1957

Session Chairman: Darwin Levine
Washington Irving High School, New York, N.Y.

9:30 A.M. –

"The Use of Isotopes in Biology Experiments in High School" – Sylvia Neivert, Bay Ridge High School, Bay Ridge, N. Y.

"Techniques in Teaching Photosynthesis" – Herbert Nestler, Franklin K. Lane High School, Brooklyn, N. Y.

"Techniques in Teaching Nutrition and Digestion" – S. Haskel, Julia Richman High School, New York, N. Y.

"Techniques in Teaching Circulation" – David Sygoda, Andrew Jackson High School, Bayside, N. Y.

"Techniques in Teaching Microbiology" – Robert Weinberger, Bronx High School of Science, Bronx, N. Y.

"Techniques in Teaching Reproduction and Development" – E. Blank, Lafayette High School, Brooklyn, N. Y.

"Techniques in Teaching Genetics" – M. Lesser, Thomas Jefferson High School, Brooklyn, N. Y.

"Techniques in Teaching Behavior" – Z. Subarsky, Bronx High School of Science, Bronx, N. Y.

"Techniques in Teaching Conservation" – T. Margaret Jamer, Public Education Association, New York, N. Y.

"Original Readings in Teaching High School Biology" – George Schwartz Forest Hills High School, Forest Hills, N. Y.

"2 x 2 Color Slides, Film Strips, and Other Photographic Aids" – H. Nagler, James Madison High School, Brooklyn, N. Y.

"New Techniques in Models, Charts, and Other Visual Aids" – Elizabeth Gray, Forest Hills High School, Forest Hills, N. Y.

12:30 P.M. –

Luncheon – Academy Building, 2 East 63rd Street, New York, N. Y.

Session Chairman: Fred Schoenberg
Board of Education of the City of New York, N. Y.

2:00 P.M. –

"The Recognition of Talent" – Paul F. Brandwein, Harcourt, Brace and Company, New York, N. Y.

"Testing for Aptitude in Science" – Charles Tanzer, Public School 101, Manhattan, New York, N. Y.

"Facilities for Developing the Talented Student" – Morris Meister, Bronx High School of Science, Bronx, N. Y.

"A Curriculum for the Talented Student in Biology" – Jerome Metzner, Bronx High School of Science, Bronx, N. Y.

"The High School Looks at the College" – A. Weckstein, Barringer High School, Newark, N. J.

"The College Looks at the High School" – Harry A. Charipper, New York University, New York, N. Y.

Discussion

Summation – R. Fuchs, Jamaica Vocational High School, Jamaica, N. Y.

ORGANIZING COMMITTEE

Harry A. Charipper, *Chairman*
Ross F. Nigrelli George Schwartz

NEW MEMBERS

NEW MEMBERS

Elected June 20, 1957

LIFE MEMBERSHIP

Nangeroni, Louis L., M.S., Physiology of Domestic Animals. Associate Professor, N. Y. State Veterinary College, Cornell University, Ithaca, N. Y.

SUSTAINING MEMBERSHIP

Aitken, W. A., D.V.M., Veterinary Medicine. Editor, American Veterinary Medical Association, Chicago, Ill.

Ball, Robert P., M.D., Radiology. Radiologist, Oak Ridge Hospital, Oak Ridge, Tenn.

Brandes, Walter W., Ph.D., Pathology. Consulting Pathologist, Roosevelt Hospital, New York, N. Y.

Gregory, Raymond, Ph.D., Internal Medicine. Professor, University of Texas, Galveston, Texas.

Hoak, Richard D., Ph.D., Chemical Research. Senior Fellow, Mellon Institute, Pittsburgh, Pa.

Illievitz, A. Bernard, M.D., Biochemistry. Medical Director, Herzl Health Centre, Montreal, Canada.

Mariani, Toni, M.A., Anatomy. Assistant Professor, Gonzaga University, Spokane, Wash.

Salomon, Mardoqueo I., M.D., Zoology. Associate, Medicine, Metropolitan Medical Center, New York Medical College, New York, N. Y.

ACTIVE MEMBERSHIP

Abramoff, Peter, Ph.D., Immunology. Instructor, Marquette University, Milwaukee, Wis.

Abrams, Adolph, Ph.D., Biochemistry. Assistant Professor, University of Colorado, Denver, Colo.

Acker, Robert Flint, Ph.D., Biochemistry. Assistant Professor, Iowa State College, Ames, Iowa.

Adams, John G., Ph.D., Pharmacology. Dean, School of Pharmacy, Duquesne University, Pittsburgh, Pa.

Adler, Frank L., Ph.D., Immunology. Associate Member, Public Health Research Institute, New York, N. Y.

Aisner, Mark, M.D., Internal Medicine. Associate Clinical Professor, Medicine, Tufts University School of Medicine, Boston, Mass.

Albert, Solomon N., M.D., Biology. Chief, Department of Anesthesiology, D.C. General Hospital, Washington, D. C.

Albrecht, William A., Ph.D., Soils and Plant Nutrition. Professor, College of Agriculture, University of Missouri, Columbia, Mo.

Albro, Helen T., Ph.D., Zoology. Head, Biology Department, Spelman College, Atlanta, Ga.

Alicante, Marcos M., Ph.D., Soil Microbiology. Director, Soils, Department of Agriculture, Manila, Philippines.

Allen, Robert D., Ph.D., Cellular Physiology. Assistant Professor, Biology, Princeton University, Princeton, N. J.

Alpert, Elmer, M.D., Internal Medicine. Director, Clinical Research, Merck Sharp & Dohme Research Laboratories, West Point, Pa.

Ames, Smith Whittier, Ph.D., Biology. Physiologist, United States Air Force, Washington, D. C.

Andy, Orlando J., M.D., Neurophysiology. Professor, University of Mississippi, Jackson, Miss.

Aptekman, Paul Myron, Ph.D., Cancer Research. Fellow, Wistar Institute of Anatomy and Biology, Philadelphia, Pa.

Arceneaux, George, Ph.D., Sugarcane Agronomy. Director, International Research Service, Christian, Miss.

Arnich, Lotte, Ph.D., Nutrition. Associate Professor, Iowa State College, Ames, Iowa.

Arrazola, Ignacio, M.D., Mathematics. Senior Resident, American Board of Radiology, Sincelejo, Colombia, S.A.

Arrigo, Sironi Dott, D.V.M., Bacteriology. Assistant, Institute Zooprofilattico, Lombarde, Italy.

Asnis, Robert E., Ph.D., Bacterial Physiology. Assistant Professor, University of Pennsylvania, Philadelphia, Pa.

Ax, Albert Francis, Ph.D., Physiology. Research Scientist, Rockland State Hospital, Orangeburg, N. Y.

Bailey, G. Howard, M.D., Bacteriology. Associate Professor, Johns Hopkins University, Baltimore, Md.

Baker, Ralph, Ph.D., Plant Pathology. Assistant Plant Pathologist, Colorado State University, Fort Collins, Colo.

Baker, Rollin H., Ph.D., Zoology. Professor, Zoology, Michigan State University, East Lansing, Mich.

Bald, John Grieve, Ph.D., Plant Pathology. Professor, University of California, Los Angeles, Calif.

Ballantyne, Donald L., Jr., Ph.D., Tissue Transplantation. Research Associate, Bellevue Medical Center, New York, N. Y.

Bancroft, Richard W., Ph.D., Physiology. Physiologist, School of Aviation Medicine, Randolph AFB, Texas.

Barclay, William Robert, M.D., Internal Medicine. Associate Professor, University of Chicago, Chicago, Ill.

Barnes, Glover W., M.A., Immunology. Assistant Cancer Research Scientist, Roswell Park Memorial Institute, Buffalo, N. Y.

Barr, C. Guinn, Ph.D., Biochemistry. Manager, Fine Chemical Technical Service, San Jose, Calif.

Barrett, James M., Ph.D., Cytology. Assistant Professor, Zoology, Marquette University, Milwaukee, Wis.

Barron, George P., Jr., Ph.D., Nutrition. Associate Professor, Pennsylvania State University, University Park, Pa.

Barrows, Charles H., Jr., Sc. D., Tissue Metabolism. Research Biochemist, National Heart Institute, Bethesda, Md.

Bauer, Robert O., M.D., Pharmacology. Associate, Roswell Park Memorial Institute, Buffalo, N. Y.

Baxter, Claude F., Ph.D., Neurochemistry. Research Associate, City of Hope Medical Center, Duarte, Calif.

Bayer, Frederick M., M.S., Zoology. Associate Curator, Division of Marine Invertebrates, Smithsonian Institution, Washington, D. C.

Bayliss, Berenice, M.S., Bacteriology. Assistant Professor, Montana State College, Bozeman, Mont.

Beam, Carl A., Ph.D., Radiogenetics. Professor, Biophysics, Yale University, New Haven, Conn.

Beams, H. W., Ph.D., Cytology. Professor, Zoology, State University of Iowa, Iowa City, Iowa.

Becker, Oscar J., M.D., Plastic Surgery. Assistant Professor, University of Illinois Medical School, Chicago, Ill.

Beeskow, Herbert Charles, Ph.D., Botany. Associate Professor, Michigan State University, East Lansing, Mich.

Bell, Helen Jean, Ph.D., Chemistry. Research Associate, University of Saskatchewan, Saskatoon, Canada.

Bell, James Milton, M.D., Medicine. Assistant Section Chief, Children's Section, Topeka State Hospital, Topeka, Kans.

Bellows, John G., M.D., Ophthalmology. Associate Professor, Northwestern University, Chicago, Ill.

Bergmann, Louis, L., M.D., Neuroanatomy. Professor, New York University, Bellevue Medical Center, New York, N. Y.

Berman, Jacob K., M.D., General Surgery. Associate Professor, Surgery, Indiana University, Indianapolis, Ind.

Berman, Shimon Elazar, M.D., Pediatrics. Chairman, Department of Pediatrics, Hebrew University, Jerusalem, Israel.

Beutner, Karl R., M.D., Neuropsychiatry. Private Practice, Vallejo, Calif.

Bever, Arley T., Ph.D., Enzymology. Associate Professor, University of Oklahoma, Oklahoma City, Okla.

Bever, Christopher T., M.D., Psychiatry. Private Practice, Washington, D.C.

Bigham, Thomas J., Rev., M.S., Psychology. Associate Professor, General Theological Seminary, New York, N. Y.

Black, Alex, Ph.D., Nutrition. Assistant Director, Pennsylvania Agricultural Experiment Station, University Park, Pa.

Blank. Isaac, General Science. President, A. Blanke & Co., New York, N. Y.

Blount, S. Gilbert, Jr., M.D., Medicine. Associate Professor, University of Colorado, Denver, Colo.

Bockus, Henry Le Roy, M.D., Gastroenterology. Professor, Department of Medicine, University of Pennsylvania, Philadelphia, Pa.

Bode, Charles E., M.S., Wheat Research. Head, Wheat Quality Laboratory, Wooster, Ohio.

Boggs, Joseph D., M.D., Pathology. Director, Children's Hospital, Chicago, Ill.

Bohl, Edward H., Ph.D., Veterinary Microbiology. Associate Professor, Ohio State University, Columbus, Ohio.

Booth, Nicholas H., M.S., Physiology. Head, Department of Physiology, Colorado State University, Fort Collins, Colo.

Bourbeau, Gerard A., Ph.D., Soil Mineralogy. Associate Professor, University of Maryland, College Park, Md.

Bove, Frank A., M.D., Diabetes Mellitus. Assistant, Philadelphia General Hospital, Philadelphia, Pa.

Bower, Albert Gordon, M.D., Acute Infectious Medicine. Chief Physician, Communicable Disease Service, Los Angeles, Calif.

Bowland, John P., Ph.D., Animal Nutrition. Associate Professor, University of Alberta, Edmonton, Canada.

Box, Edith Darrow, Sc.D., Biology. Associate Professor, University of Texas Medical Branch, Galveston, Texas.

Boyd, Eugene Stanley, Ph.D., Drug Metabolism. Pharmacologist, University of Rochester, Churchville, N. Y.

Boynton, Damon, Ph.D., Pomology. Professor, Cornell University, Ithaca, N. Y.

Brady, Allan J., Ph.D., Biophysics. Honorary Instructor, University of Washington, Seattle, Wash.

Brady, Edward S., Ph.D., Pharmaceutical Chemistry. Director, Research, Horton and Converse, Los Angeles, Calif.

Bragg, Robert L., M.D., Psychiatry. Resident, Medfield State Hospital, Harding, Mass.

Brasfield, Richard D., M.D., Cancer Therapy. Assistant Surgeon, Memorial Hospital, New York, N. Y.

Brewer, N. R., Ph.D., Laboratory Animals. Research Associate, University of Chicago, Chicago, Ill.

Brink, R. A., Sc.D., Genetics, Botany. Professor, University of Wisconsin, Madison, Wis.

Brody, Theodore M., Ph.D., Pharmacology. Assistant Professor, University of Michigan, Ann Arbor, Mich.

Brookbank, John W., Ph.D., Experimental Embryology. Assistant Professor, University of Florida, Gainesville, Fla.

Brophy, Daniel F., M.D., Psychology. Dean of Students, The City College of New York, New York, N. Y.

Brown, G. Malcolm, M.D., Cold Acclimatization. Professor, Medicine, Queen's University, Kingston, Ont., Canada.

Brozek, Josef, Ph.D., Human Biology. Professor, University of Minnesota, Minneapolis, Minn.

Brumfield, Helene Paine, Ph.D., Immunology. Research Fellow, University of Minnesota, Minneapolis, Minn.

Brummett, Anna Ruth, Ph.D., Experimental Embryology. Assistant Professor, Carleton College, Northfield, Minn.

Buchwald, Nathaniel A., Ph.D., Neurophysiology. Physiologist, Southwest Louisiana Hospital, Mandeville, La.
Buckley, Joseph P., Ph.D., Pharmacology. Associate Professor, University of Pittsburgh, Pittsburgh, Pa.
Bullis, Kenneth L., D.V.M., Veterinary Science. Head, Department of Veterinary Science, University of Massachusetts, Amherst, Mass.
Burnett, George Wesley, D.D.S., Microbiology. Chief, Dental Research, Walter Reed Army Institute, Washington, D.C.
Burrows, William, Ph.D., Immunology. Professor, University of Chicago, Chicago, Ill.
Buxeda, Roberto, M.D., Ophthalmology. Assistant Professor, University of Puerto Rico, Santurce, Puerto Rico.
Buxton, Robert W., M.D., Surgery. Professor, University of Maryland, Baltimore, Md.
Byatt, Pamela H., Ph.D., Virology. Research Virologist, University of California, Los Angeles, Calif.
Byham, Bruce Bodine, M.S., Pathology. Research Associate, Merck Sharpe & Dohme, West Point, Pa.
Calabresi, Massimo, M.D., Physiology. Chief, Cardiovascular Section, Veterans Administration Hospital, West Haven, Conn.
Cameron, John, M.D., Anatomy. Retired, Bournemouth, England.
Campbell, James B., M.D., Neurosurgery. Assistant Professor, Neurological Institute, New York, N. Y.
Cardiff, Ira D., Ph.D., Biology. President, Washington Dehydrated Food Company, Yakima, Wash.
Cason, Dick, M.D., Biology. Private Practice, Hillsboro, Texas.
Cassebaum, William H., M.D., Surgery. Attending Surgeon, Roosevelt Hospital, New York, N. Y.
Catz, Boris, M.D., Clinical Research. Assistant Clinical Professor, Medicine, University of California, Los Angeles, Calif.
Cavalieri, Liebe F., Ph.D., Physical Chemistry. Associate Professor, Sloan-Kettering Institute, New York, N. Y.
Cogan, Herbert J., A.B., Anthropology. Financial Counsel, Brooklyn, N. Y.
Cohen, Arthur I., Ph.D., Physiology. Research Associate, Children's Cancer Research Foundation, Boston, Mass.
Cohen, Morris, Ph.D., Virology. Electron Microscopist, St. Joseph Hospital, Burbank, Calif.
Cole, Johnathen O., M.D., Psychiatry. Chief, Psychopharmacology Service Center, National Institute of Mental Health, Bethesda, Md.
Cooley, Robert Nelson, M.D., Radiology. Professor, Department of Radiology, University of Texas, Galveston, Texas.
Cowan, Robert L., Ph.D., Animal Nutrition. Associate Professor, Pennsylvania State University, University Park, Pa.
Crabb, Edward Drane, Ph.D., Cancer Research. Professor, University of Colorado, Boulder, Colo.
Craig, Peter H., D.V.M., Biology. Research Institute, University of Pennsylvania, Drexel Hill, Pa.
Craig, Robert Leslie, Ph.D., Mechanisms of Ascites Accumulation. Clinical Research Investigator, G. D. Searle, Inc., Chicago, Ill.
Cutting, W. C., M.D., Pharmacology. Professor, Stanford University, Stanford, Calif.
Dale, C. N., D.V.M., Veterinary Bacteriology. Veterinarian, U. S. Department of Agriculture, Beltsville, Md.
Dawe, Albert R., Ph.D., Physiology. Biologist, Office of Naval Research, Chicago, Ill.
Deere, Charles Joseph, M.D., Internal Medicine. Assistant Professor, University of Tennessee, Memphis, Tenn.
Delmar, Geza Szabados, Ch. Eng., Chemistry. President, Delmar Chemicals Ltd., Lachine, Canada.
Doering, Milan F., M.A., Psychology. Fellow, Western Psychiatric Institute, Pittsburgh, Pa.
Dougherty, Robert W., M.S., Physiology. Professor, Cornell University, Ithaca, N. Y.

Dove, Walter E., Sc.D., Insecticides. Entomologist, Fairfield Chemical Division, Baltimore, Md.

Eberts, Floyd S., Jr., Ph.D., Biochemistry. Research Associate, Upjohn Company, Kalamazoo, Mich.

Fand, Irwin, Ph.D., Cytophysiology. Research Scientist, Creedmoor Institute, Queens Village, N. Y.

Fillios, Louis Charles, Sc.D., Nutritional Biochemistry. Research Associate, Harvard School of Public Health, Boston, Mass.

Freedlender, Arthur Elliott, Protein Chemistry. Student, Harvard University, Jamaica Plain, Mass.

Gonzaga, Arcadio C., Ph.D., Veterinary Physiology. Professor and Head, University of the Philippines, Quezon City, Philippines.

Gordon, I. Bradley, B.A., Connective Tissue Disorders. Student, Columbia University, New York, N. Y.

Grainger, E. H., Ph.D., Marine Biology. Associate Scientist, Arctic Unit, Fisheries Research Board of Canada, Montreal, Canada.

Gray, Louise F., M.S., Animal Nutrition. Biochemist, U.S. Plant, Soil & Nutrition Laboratory, Ithaca, N. Y.

Greene, Harry S. N., M.D., Medicine. Cancer Research. Chairman, Department of Pathology, Yale University, New Haven, Conn.

Greiff, Donald, Sc.D., Virology. Professor, Pathology, Marquette University, Milwaukee, Wis.

Griswold, Robert L., Ph.D., Physiology. Chief, Department of Biochemistry, Walter Reed Army Institute of Research, Washington, D.C.

Gunn, Charles Kenneth, Ph.D., Biology. Superintendent, Dominion Experimental Fur Ranch, Summerside, Canada.

Gustafson, Philip F., M.S., Physics. Assistant Physicist, Argonne National Laboratory, Berwyn, Ill.

Guthrie, James Erwin, D.V.M., Veterinary Science. Associate Director, Research, Hess & Clarke, Inc., Ashland, Ohio.

Hachemeister, Charles A., D.E.E., Teaching. Professor, Electrical Engineering, Polytechnic Institute of Brooklyn, Brooklyn, N. Y.

Hall, Harvey, Ph.D., Physics. Director, Operation Research Division, Office of Naval Research, Fairfax, Va.

Haller, Wolfgang, Ph.D., Physical Chemistry. Associate Professor, University of Toledo, Toledo, Ohio.

Halwer, Murray, Ph.D., Analytical Chemistry. Chief, Development Group, Lederle Laboratories, Pearl River, N. Y.

Ham, William Taylor, Ph.D., Biophysics. Chairman, Department of Biophysics, Medical College of Virginia, Richmond, Va.

Hannan, Charles Kevin, Ph.D., Microbiology. Assistant Professor, University of Manitoba, Winnipeg, Canada.

Hannum, John Andrew, Ph.D., Physical Science. President, Hefco Laboratories, Inc., Detroit, Mich.

Hansen, Ralph Holm, Ph.D., Polymerization. Member, Technical Staff, Bell Telephone Laboratories, Murray Hill, N. J.

Hanson, William Ellum, Ph.D., Geochemistry. Assistant Fellow, Mellon Institute, Pittsburgh, Pa.

Hansteen, Henry B., Ph.D., Electrical Engineering, Professor, The City College of New York, New York, N. Y.

Harber, William I., Ph.D., Polymers. Chemical Consultant, Wilson Junior College, Chicago, Ill.

Harned, Roger Lee, M.S., Microbiology. Group Leader, Commercial Solvents Corporation, Terre Haute, Ind.

Harrell, Bryant, Ph.D., Organic Chemistry. Assistant Professor, College of William & Mary, Williamsburg, Va.

Harris, Gordon M., Ph.D., Physical Chemistry. Professor, University of Buffalo, Buffalo, N. Y.

Harris, James T., Jr., Ph.D., Chemistry. Retired. Spartanburg, S. C.

Harris, Warren W., B.S., Electron Microscopy. Head, Electron Microscopy, Oak Ridge, Tenn.

Harris, William M., Ph.D., Physics. Physicist, Zenith Radio Research Corporation, Redwood City, Calif.

Harrold, Orville G., Jr., Ph.D., Mathematics. Professor, University of Tennessee, Knoxville, Tenn.

Hart, Hiram, Ph.D., Biophysics. Assistant Professor, The City College of New York, New York, N. Y.

Hartman, Richard E., Ph.D., Biophysics. Chief, Biophysics Section, Walter Reed Army Institute, Washington, D. C.

Hartung, Walter H., Ph.D., Chemistry. Professor, Medical College of Virginia, Richmond, Va.

Harwood, H. James, Ph.D., Chemistry. Manager, Central Research Department, Armour & Co., Chicago, Ill.

Haven, Alfred C., Jr., Ph.D., Chemistry. Division Head, Jackson Laboratory, E. I. duPont de Nemours & Co., Inc., Wilmington, Del.

Hayner, Lucy Julia, Ph.D., Physics. Associate Professor, Columbia University, New York, N. Y.

Heiberger, Philip, Ph.D., Chemistry. Research Chemist, E. I. duPont de Nemours & Co., Inc., Wilmington, Del.

Heidenreich, Robert D., M.S., Microscopy. Staff, Bell Telephone Laboratories, Inc., Murray Hill, N. J.

Hein, Richard E., Ph.D., Chemistry. Fellow, Mellon Institute, Pittsburgh, Pa.

Heines, Virginia, S.C.N., Ph.D., Chemistry. Professor, Nazareth College, Louisville, Ky.

Heinrich, Edwin P., Ph.D., Physics. Headmaster, St. Johns Country Day School, Orange Park, Fla.

Heller, Wilfried. Ph.D., Chemistry. Professor, Wayne State University, Detroit, Mich.

Henry, Warren E., Ph.D., Physics. Physicist, Naval Research Laboratory, Washington, D. C.

Herman, Robert, Ph.D., Physics. Consulting Physicist, General Motors Corporation, Detroit, Mich.

Herold, Edward W., M.S., Electronics. Director, Electronic Research Laboratory, RCA Laboratories, Princeton, N. J.

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Elected August 15, 1957

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Marcelli, Joseph Frederick, Ph.D., Organic Chemistry. Associate Professor, Hudson Valley Technical Institute, Troy, N. Y.
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Milles, George, M.D., Pathology. Director, Laboratories, Augustana Hospital, Chicago, Ill.
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Newhall, Allan G., Ph.D., Plant Pathology. Staff, Chiriqui Land Company, Almirante, Republic of Panama.
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Powell, Sheppard T., Engineering. Consulting Engineer, Baltimore, Md.
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Schlossman, Stuart Franklin, B.A., Pathology. Medical Student, New York University, New York, N. Y.
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Elected September 26, 1957

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Rich, George R., Dr. Eng., Hydraulic Engineering. Director, Charles T. Main, Inc., Boston, Mass.
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ACTIVE MEMBERSHIP

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